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Postgraduate Medicine

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Surgical Treatment of Inguinal Hernia

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THE three most common surgical conditions that we are called upon to handle are appendicitis, hernia, and some form of trauma. Yet, I think that up until eight or nine years ago, interest in hernia had somewhat waned in behalf of problems such as the postgastrectomy syndromes and difficulties, and the great advances in physiology as they relate to major operations, and we have tended to neglect our hernia friend who is with us always. We have had perhaps the same attitude as a very charming friend of mine who was 75 years of age and was invited one day to join the Birth Control League. She said, "What! Me? At 75?" And they said, "Yes, sure, we would like you to sign up."

"Well," she said, "all right, I'll sign up." So she signed up and wrote after her name, "Interested but not active."

On the other hand, we have had very definitely in the hernia problem new interest aroused by the experimental work and particularly the anatomic research that has been conducted in the last eight or nine years, especially stimulated by the work of McVey and Anson. Therefore, I wish to discuss with you some of the newer developments in the surgical treatment of hernia which might prove of interest and perhaps of value.

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Presented October 31, 1949, before the meeting of the Interstate Postgraduate Medical Association of North America, Philadelphia, Pennsylvania.

If an operation for hernia is contemplated, it is important to know the background of procedures that have been used and suggested heretofore for the cure of this lesion. It would be well, then, to give a short historical review of the development of the operative steps and procedures that must be kept in mind when a herniotomy is to be performed (Table 1).

You will remember that Bassini and Halsted began their work almost simultaneously between 1888 and 1890. Bassini described for the closure of the inguinal canal the use of the internal oblique sutured to Poupart's ligament beneath the cord. This was a distinct anatomic advance over previously suggested procedures, which were comparatively simple and consisted, surprisingly enough, merely of suture of the internal or external ring.

Halsted, about the same time, but not reported until 1893, not only used the internal oblique sutured to Poupart's ligament beneath the cord, but also sutured the *external* oblique beneath the cord, the cord being transplanted above both of these structures. For the proposal of nontransplantation of the cord, credit has usually been assigned to Ferguson (1899). However, Dr. W. B. Coley in 1895 had suggested its use, and Dr. Halsted independently advised leaving the cord in situ, in a paper published in 1903.

Andrews, in 1895, proposed overlapping of the aponeurosis of the external oblique, one flap beneath the cord and one over the cord. McArthur, in 1901, reported the use of pedicled strips of fascia of the external oblique for suture

WILLIAM L. ESTES, JR.

TABLE 1

BACKGROUND OF PROCEDURES USED IN TREATMENT OF INGUINAL HERNIA

1. BASSINI—1890
   Internal oblique sutured to Poupart's ligament (inguinal) beneath cord.
2. HALSTED—1893
   Cord transplanted above external oblique.
3. W. B. COLEY—1895
   FERGUSON—1899
   HALSTED—1903
   Nontransplantation of cord.
4. ANDREWS—1895
   Overlapping aponeurosis of external oblique.
5. MC ARTHUR—1901
   Pedicled strips of fascia of external oblique for suture material.
6. HALSTED—1903
   BLOODGOOD—1919
   Rectus sheath and rectus muscle.
7. SEELIG—1923
   Fascia to fascia—strongest closure.
8. GALLIE AND LE MESURIER—1923
   Strips of fascia lata for suture.
9. WANGENSTEEN—1934
   Pedicled flap of fascia lata.
10. MC FEE—1940
    Transplantation of cord to femoral canal.
11. MC VEY AND HARKINS—1941
    Suture of conjoined tendon to Cooper's ligament in Bassini type operation.

material. And in 1903, Halsted described a further variation in procedure whereby the rectus sheath and the rectus muscle could be used, when the structures of the canal were thin and weak, instead of the internal oblique or conjoined tendon. Bloodgood later (1919) elaborated on the use of the rectus muscle.

In 1923, Seelig demonstrated that suture of fascia to fascia is the strongest closure; fascia to muscle will give firm healing, but fascia to fascia is stronger.

We next have Gallie and Le Mesurier with their well-known work of using strips of fascia lata for suture. In 1934 Wangensteen advised a pedicled flap of fascia lata reflected from the thigh up to the inguinal area in cases where there was marked weakness of the structures. McFee tried to meet this problem by transplantation of the cord to the femoral canal. Finally the most recent proposal that has given the greatest impetus to renewed interest in the operation for hernia is the work of McVey (1944) backed up by Harkins. McVey suggested, in the Bassini type of closure, that the conjoined tendon should be sutured to Cooper's ligament, not the inguinal ligament, because the transversalis arises from or is normally attached to Cooper's ligament rather than the inguinal ligament.

Up to 1940, the standard types of operation were largely those of Bassini, of Halsted—the transplantation of the cord above the internal and external oblique—or of Ferguson—or modifications of these.

END results reported up to 1940 are summarized in Table 2. Of the recurrence rates for the Bassini type operation, Gibson's is the most creditable. The other rates of recurrence are quite high for this frequently performed operation. Halsted type recurrence rates were reasonably high also in the treatment of indirect hernia. In the treatment of direct hernia there is an *extraordinarily* high percentage of recurrence. The need for some procedure that would give better results seems definitely indicated.

In planning an operation for inguinal hernia, knowledge of the pathology and variations in anatomy that may be found is essential. The chief elements in the pathology are largely: (1) the various types of inguinal hernia, and (2) the defects that may be present in the structures that are used for repair. These defects are very

TABLE 2

END RESULTS IN TREATMENT OF INGUINAL HERNIA REPORTED UP TO 1940

AUTHOR	RECURRENCE RATE, PER CENT	
	BASSINI	HALSTED
Indirect Hernia		
Coley	8.7	
Erdman	3.15	
Gibson	1.3	
Taylor		5.6
Direct Hernia		
Coley	16.4	
Erdman	16.6	
Shelley	14.8	
Gibson	6.5	
Taylor		18.08

TABLE 3

TYPES OF INGUINAL HERNIA

ETIOLOGY
1. Traumatic
   Stress Strain
2. Anatomic

PATHOLOGY
1. Indirect—congenital
2. Direct
3. Direct indirect—sliding
4. Strangulation—incarceration

important to recognize, and demand careful treatment if high percentage of recurrence is to be avoided.

1. There are three types of inguinal hernia (Table 3): (*a*) the direct, (*b*) the indirect, and (*c*) the direct-indirect or the sliding hernia. This latter is the type in which there is no preformed sac, but the nearby peritoneum and tissues attached to it—on the left these may be the sigmoid and its mesentery—slide down through a large internal ring and we have what is called a direct type of indirect hernia. All types of hernias may be incarcerated or strangulated. The difference between the indirect and the direct sac, as you know, is determined by their relation to the deep epigastric vessels; that which is lateral to the vessels is an indirect sac, and the direct hernias are those in which the sac is medial to the epigastric vessels.

2. The defects that must be recognized are: (*a*) an over-large internal ring; (*b*) a small direct hernia in association with an obvious larger indirect hernia; (*c*) weaknesses, openings, or defects of and in the transversalis muscle, the posterior wall of the inguinal canal. These are all important to recognize because they must be corrected by the operative procedure.

OPERATIVE TECHNIC

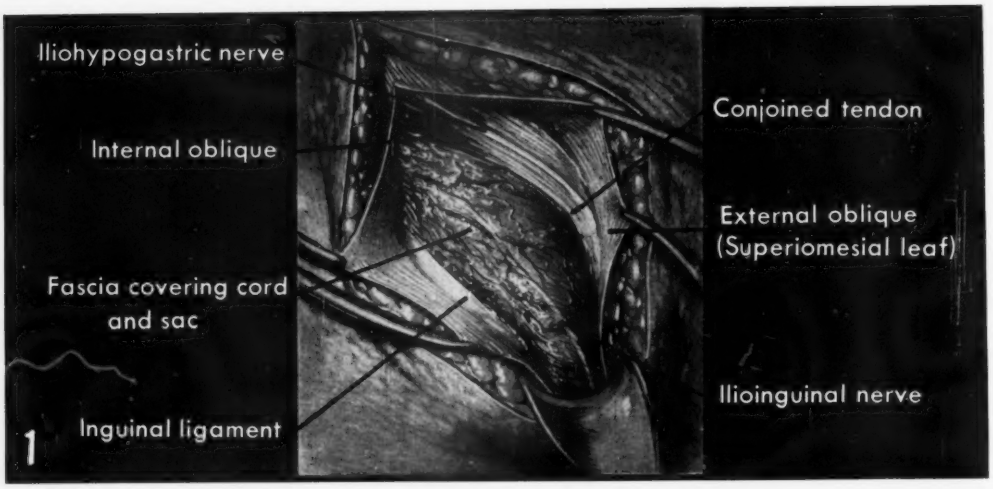
THERE are certain essentials in operative technic that must be observed if accurate, solid healing is to be obtained—complete relaxation of muscles is a sine qua non. Therefore, spinal anesthesia is very definitely to be preferred. Fine ties should be used for hemostasis; also sharp dissection; gentle handling of tissues, nonabsorbable sutures, either silk or cotton (we favor cotton), and suturing without tension.

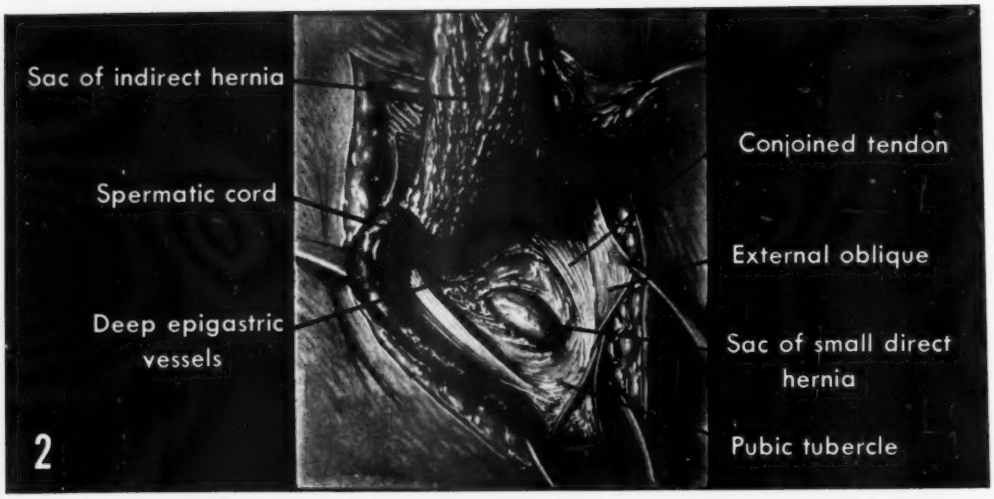
There are three operations for inguinal hernia that can be recommended: (1) a Bassini type of operation, using Cooper's ligament in the first layer of sutures; (2) an operation in which the rectus sheath and/or rectus muscle are used in the closure, and finally (3) an atypical Halsted type of operation for the few and rare hernias where the structures will be so weak that tantalum mesh or variations from customary technic may have to be employed.

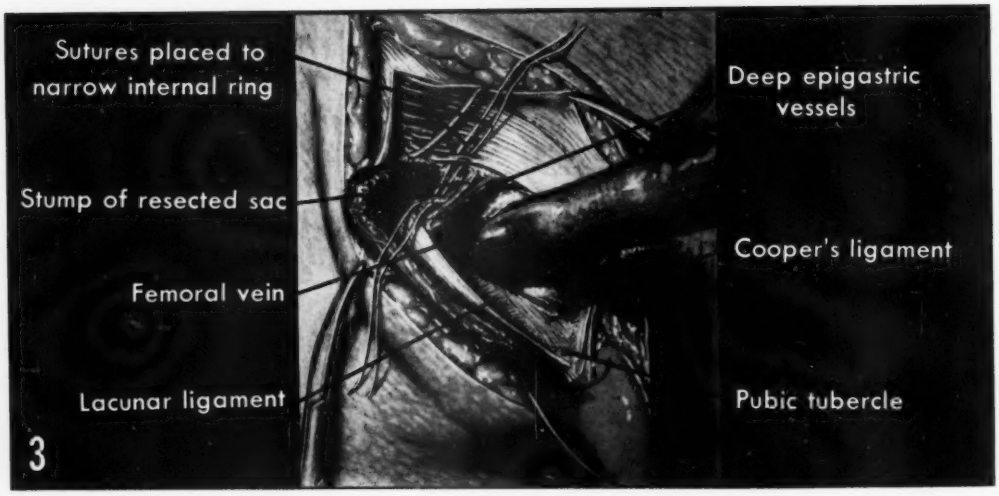
In these, particularly, the old Halsted type operation where all tissues are used to strengthen the inguinal canal, the cord brought out directly at the level of the internal ring, is to be favored, but the addition of tantalum mesh gauze to strengthen thinned out or weakened tissue, I believe is a very definite asset to our armamentarium for this type of hernia. However, I would like to concentrate upon and describe in detail the first two types of operations which we have found most useful and successful in handling this inguinal hernia problem.

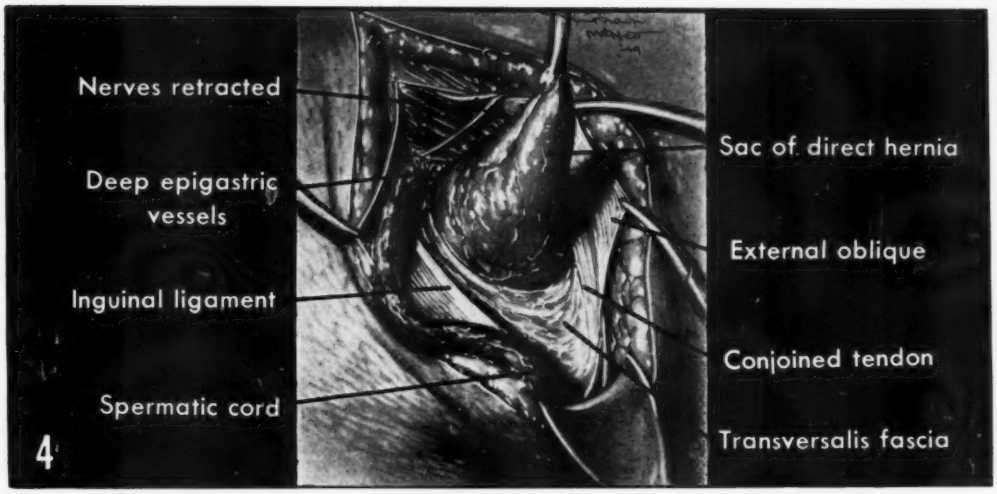
The incision is made a little oblique to the inguinal or Poupart's ligament, 8 to 10 cm. in length (Figure 1). The external oblique is split over the middle of the bulge of the cord lying just beneath and laid open. Beneath, is the cord swollen by the sac; the ilio-inguinal nerve is on the surface of the cord, and the ilio-hypogastric is located medially; also seen are the internal oblique, the sheath of the rectus, and the conjoined tendon where it joins the transversalis. The lower angle of the wound should expose the pubic tubercle which must always be recognized. The pubic tubercle should be right within the lower angle of the wound to have adequate exposure (Figure 2).

Your attention should be called to the little swelling which is a small direct hernia, in addition to the obvious or indirect hernia (Figure

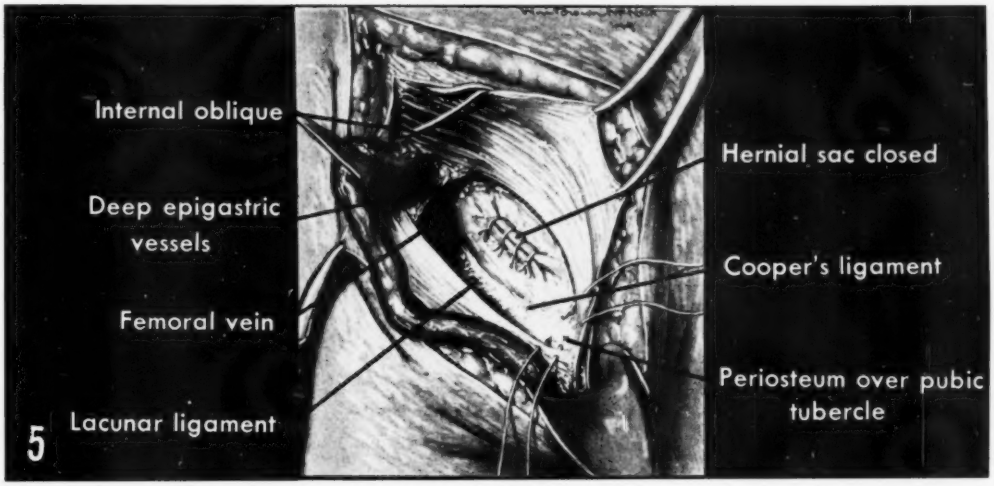

Iliohypogastric nerve
Internal oblique
Fascia covering cord and sac
Inguinal ligament
1
Conjoined tendon
External oblique (Superiomesial leaf)
Ilioinguinal nerve


Sac of indirect hernia
Spermatic cord
Deep epigastric vessels
2
Conjoined tendon
External oblique
Sac of small direct hernia
Pubic tubercle

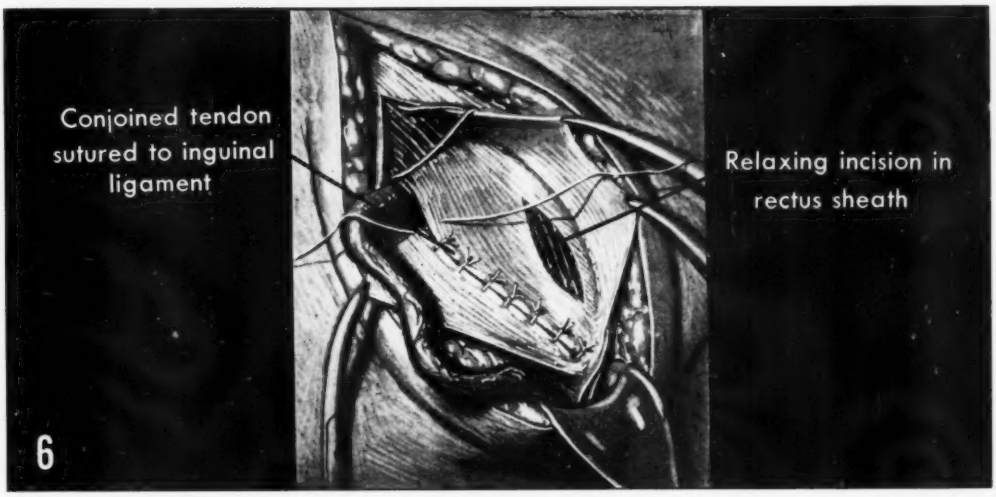

Sutures placed to narrow internal ring
Stump of resected sac
Femoral vein
Lacunar ligament
3
Deep epigastric vessels
Cooper's ligament
Pubic tubercle


Nerves retracted
Deep epigastric vessels
Inguinal ligament
Spermatic cord
Sac of direct hernia
External oblique
Conjoined tendon
Transversalis fascia

4


Internal oblique
Deep epigastric vessels
Femoral vein
Lacunar ligament
Hernial sac closed
Cooper's ligament
Periosteum over pubic tubercle

5


Conjoined tendon sutured to inguinal ligament
Relaxing incision in rectus sheath

6

2). If these defects are not looked for they may easily be missed and a very definite factor in recurrences allowed to persist.

In this case, the large fatty mass attached to the cord has been freed. This is the so-called sliding type of hernia. The cord has been retracted, and the deep epigastric vessels are visible.

A SLIDING hernia should be recognized by its wide base. In ordinary indirect hernia the base is narrow. A sac with a narrow base is ligated at its neck, using high ligation, and resected, and then its stump will retract well up beneath the muscle. However, in a sliding hernia, seldom is there need to do anything to the sac. There is really no sac. Occasionally there is excessive peritoneum that you can get rid of, but that is the exception. This is the type of hernia that usually needs no resection. It is nothing but abdominal contents prolapsed through or forced through an opening, or defect in the muscle, and it should be restored again to the abdomen.

The next procedure is to close the large internal ring, which such a hernia almost invariably has (Figure 3). It is closed snugly about the cord; it must not be too tight. The sutures go through the edge of the transversalis or the internal oblique and the transversalis on the lateral side. Interrupted cotton sutures or interrupted nonabsorbable sutures are favored. The test for the closure's not being too tight is to be able to get an index finger down through the internal ring after this suture has been tightened. Cooper's ligament and the pubic tubercle should now be identified and the inguinal ligament as well.

To demonstrate a direct hernia Figure 4 is presented. The ilio-inguinal nerve in this case has been retracted over upon the medial surface of the external oblique, to isolate better the large direct hernia.

The closure of this hernia is accomplished in the same way as for a sliding hernia (Figure 5). Very rarely, these will be small with a true sac, have a narrow neck, and can be resected. In most instances, however, they have no sac. They are similar to sliding hernias; i.e., a direct hernia is nothing but abdominal and peritoneal contents that have slid down through an opening in the transversalis muscle and it should be restored to where it came from—the abdominal cavity. It is largely properitoneal fat with peritoneum and in many instances the bladder. Here is where you will come to grief if you try to resect. These direct hernias must be freed from all attachments and restored to the abdominal cavity without resection. Then the opening or hernial orifice in the transversalis is closed with interrupted cotton sutures (Figure 5).

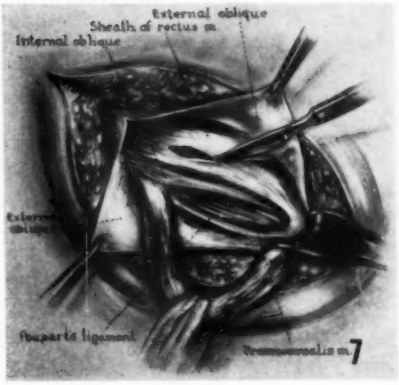
For closure of the inguinal canal the two methods of suture that have been found most valuable will be described in detail and may be used for both direct or indirect hernias: (1) the Bassini type where the conjoined tendon is sutured to Cooper's ligament, and (2) that in which the rectus sheath or the rectus muscle—or both—are used instead of the conjoined tendon.

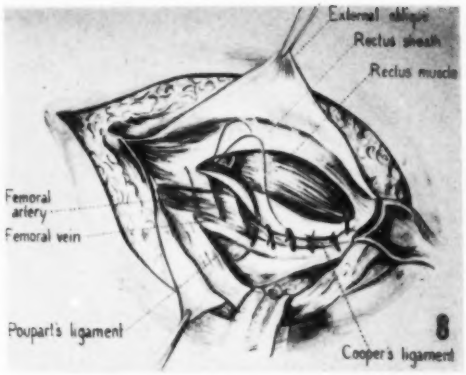
Bassini type—The first suture passes through the junction of the conjoined tendon with the pubic tubercle, through the periosteum of the tubercle, and the first fibers of the inguinal ligament. The next suture, and all the others up to the level of the femoral vessels, pass through the conjoined tendon, Cooper's ligament, and through the edge of the inguinal ligament, so that the conjoined tendon is brought down to Cooper's and the inguinal ligaments. This maneuver also closes the femoral canal area and prevents a later femoral hernia. However, beyond and lateral to the femoral vessels, the sutures are inserted, from the conjoined tendon or internal oblique to the inguinal ligament only, up to the level of the internal ring, to fit snugly against the cord.

In Figure 6 the sutures are tied, and the last suture has just been inserted showing the final suture through the internal oblique and the inguinal ligament alone. Often upon completion of this layer of sutures, some tension is observed. To relieve this tension an oblique relaxing incision high in the rectus sheath, originally suggested by Halsted, revived by Rienhoff and numerous others more recently, may be used.

The operation is then concluded by dropping the cord and nerve back over the suture line and internal oblique, and closing the external oblique over the cord.

The rectus sheath type of closure (Figures 7-12)—Twenty to twenty-five years ago I discovered that I was having entirely too many recurrences from the old Halsted and Bassini types of operation, so turned to the use of the rectus sheath and overlapping of the fascia of the external oblique for a more secure restoration of the inguinal canal.



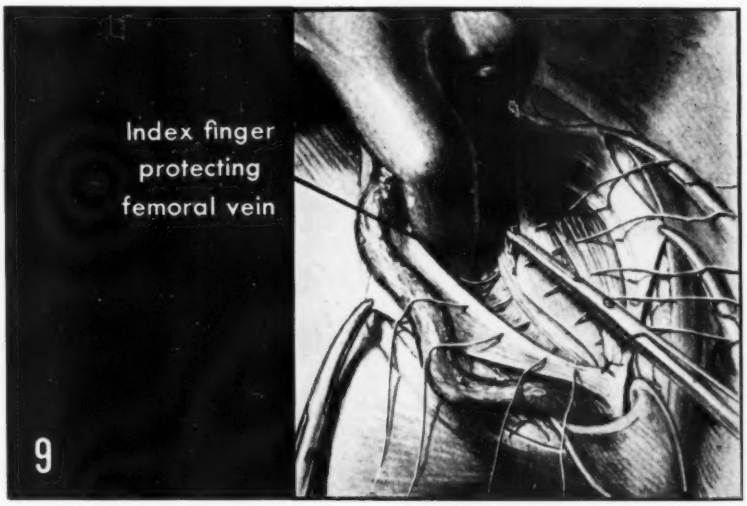


An incision is made in the sheath of the rectus about a centimeter from the margin of the rectus muscle (Figure 7). It is elliptical and curves a little toward the internal ring and down toward the pubic tubercle (Figure 8).

At first the flap of rectus sheath thus isolated was rotated and reflected laterally and sutured to the inguinal ligament. However, we found that in reflecting this flap often a definite weakness is left at the lateral margin of the muscle, where frequently the posterior sheath may be very thin or nonexistent. Also suture to Cooper's ligament is to be preferred to the use of the inguinal ligament.

Therefore, what is used now for the first line of sutures, is a four-ply apposition (Figure 9). The first suture passes through the attachment of the pyramidalis and rectus muscle to the pubic tubercle through the periosteum of the pubic tubercle and the beginning of the inguinal ligament. The next two or three sutures pass through the tendinous margin of the rectus muscle, take a bite in the rectus sheath, and pick up the margin of Cooper's ligament and the margin of the inguinal ligament. These should be placed at first without tying because of the difficulty of inserting a suture accurately in Cooper's ligament and of damage to the femoral vessels unless accurate visualization is maintained. Sutures through Cooper's ligament extend only to the femoral vessels (Figure 9).

To avoid damage to them, the left index fin-



ger may be kept on the femoral vein and the last suture next to the femoral vein is never nearer than a centimeter to that finger. It will be noted that when the suture which passes through the tendinous margin of the muscle, the leaf of the sheath of the muscle, Cooper's ligament, and then the inguinal ligament, is tied, there is *broad surface* to *broad surface*, and not simply *point* contact. Likewise, it is largely a fascia-to-fascia closure with the exception of the tendinous margin of the muscle.

The final sutures, lateral to and over the femoral vessels, up to the internal ring, are inserted through the tendinous margin of the rectus, or the internal oblique, the sheath of the rectus, and the inguinal ligament only, as in the older types of operation (Figure 10). The final suture must not be too tight or placed too near the cord, because that will cause constriction of the vessels of the cord and lead to atrophy of the testicle.

The next layer of sutures brings the lateral leaf of the external oblique (attached to the inguinal ligament) over to the medial margin of the sheath of the rectus, to form a new anterior sheath for the rectus.

Figure 11 shows the sutures being placed. The structures are thus superimposed to form a new double floor for the inguinal canal, but the suture lines are not superimposed, which gives added strength.

The cord is then dropped back over this new rectus sheath and the medial flap of the external oblique is brought over the cord and sutured to the inguinal ligament or to the fascia just medial to the inguinal ligament to form a new roof of the canal (Figure 12). A few interrupted sutures are placed in the deep layer of the superficial fascia and the skin is closed.

The postoperative care of these patients is very simple. They are up the day after the operation. Many of us of the olden days might shudder at that idea, but we must realize that early ambulation really stimulates wound healing and can begin safely the day following operation. The patient is hospitalized for about a week. Bilateral hernias may be repaired at the same time. Postoperatively, patients are permitted to go back to work usually within six to eight weeks; for extremely heavy, arduous labor, the period of time should be three months, but there is no permanent restriction on activity.

I want to conclude briefly by showing you the end results we have obtained, particularly with this last type of operation that I have just demonstrated. The total number of hernias examined in our first follow-up study was 416, indirect, direct, and recurrent (Table 4).

TABLE 4
TOTAL CASES EXAMINED IN FIRST FOLLOW-UP STUDY

Total cases	394
Bilateral	22
Total hernias	416
Indirect	317
Direct	80
Recurrent	19

The age incidence is of no particular value except to show that there is an ample number of older people included in this group (Table 5).

TABLE 5
AGE INCIDENCE

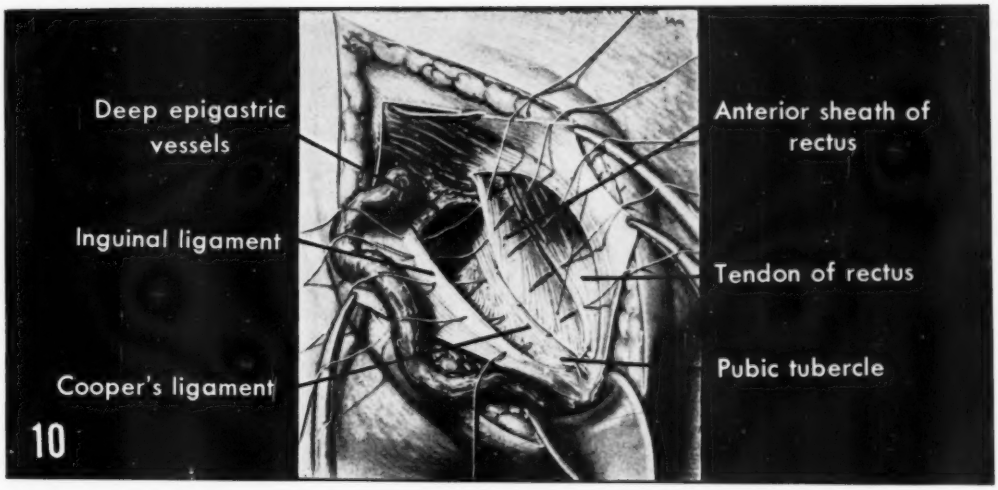
AGE	NUMBER
Under 20	40
20 to 30	95
30 to 40	85
40 to 50	100
50 to 60	50
Over 60	24
	394

We were able to follow 359 of the patients. These were all personal follow-ups. None reported by letter; every one was examined, by me or one of my associates (Table 6). Tables 7 and 8 show the follow-up results and end results.

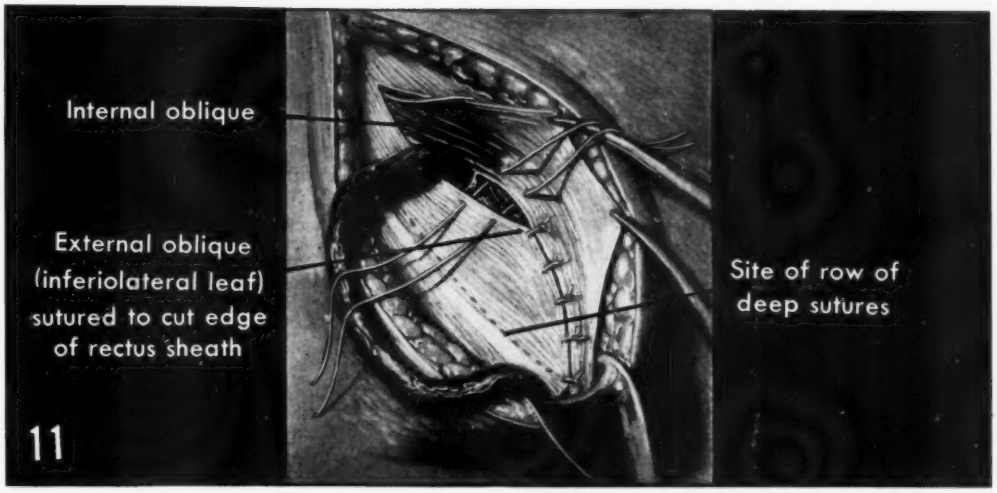
TABLE 6
FOLLOW-UP RECORD

Total cases	394	
Hernias examined	359	(including bilateral)
Examinations made	339	(86 per cent)
Records unobtained	55	(14 per cent)
Cases followed 10 to 14 years	21	
Cases followed 5 to 10 years	63	
Cases followed 1 to 5 years	74	
Cases followed one year or under	181	

Of the postoperative complications, I particularly want to call attention to atrophy of the testicle. In this first group there were 12 cases or 3.34 per cent. This is too high. We didn't


Deep epigastric vessels
Inguinal ligament
Cooper's ligament
Anterior sheath of rectus
Tendon of rectus
Pubic tubercle

10


Internal oblique
External oblique (inferiolateral leaf) sutured to cut edge of rectus sheath
Site of row of deep sutures

11

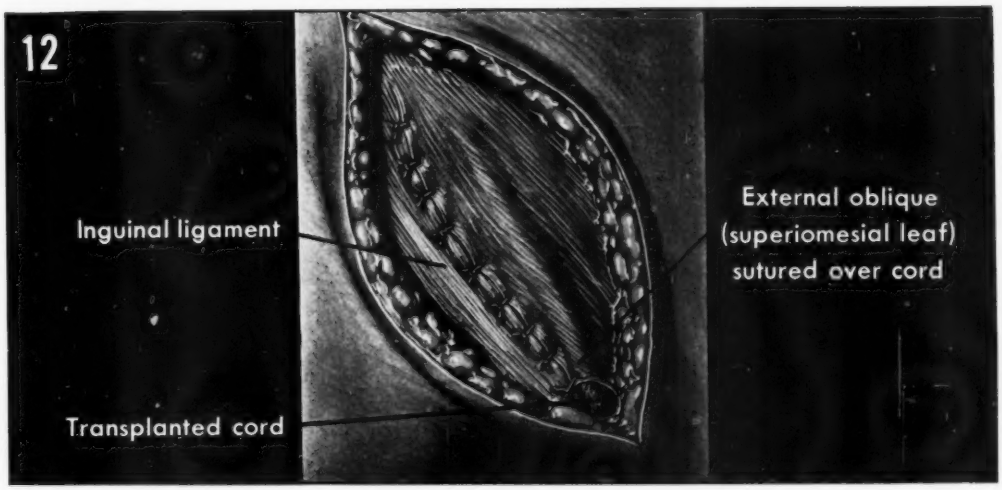

12
Inguinal ligament
Transplanted cord
External oblique (superiomesial leaf) sutured over cord

TABLE 7
FOLLOW-UP RESULTS

	TOTAL HERNIAS	FOLLOW-UP EXAMINATION OBTAINED	RECURRENCE	
			Number	*Per Cent**
Indirect	317	271	1	0.4
Direct	80	72	2	2.77
Recurrent	19	16	2	12.5

*Hernias examined.

TABLE 8
END RESULTS OF FOLLOW-UP

Recurrences		
Total hernias	416	
Recurrence (postoperative)	5	(1.2 per cent)
Postoperative Complications		
Wound infections	3	
Delirium	1	
Strangulated	2	
Atrophy of testicle	12	(3.34 per cent*)
Hydrocele	1	

*Hernias examined.

realize this possibility until we began our end result studies. It is due to too tight suturing, chiefly at the internal ring, and we have been able to reduce the incidence of testicular atrophy materially by being careful not to suture too snugly at either the external or the internal ring.

This first study was not completely satisfactory because about one-half of the cases we had been able to follow for only a year, so we did first a three-year and now finally a five-year study. Statistics for a full three-year follow-up show end results as listed in Table 9. In this total group there were 25 sliding hernias with no recurrence. This would seem to indicate that the rectus sheath rectus muscle closure method is a satisfactory operation for the sliding type of hernia.

TABLE 9
RESULTS OF THREE-YEAR FOLLOW-UP

		RECURRENCE	
		Number	*Per Cent*
Indirect	(119)	1	0.84
Direct	(56)	3	5.3
Recurrent	(11)	0	
TOTAL	186	4	2.1
Sliding hernia	(25)	0	

Table 10 is a five-year study of Cooper's ligament repair. These 149 cases were observed for five years or more; some of these were followed for seven years. This group unfortunately is small because we have only used this type of repair since 1943, and therefore have only been able to include the cases operated upon in a two or three-year period. Of this group, there were 90 indirect hernias with one recurrence. We did have 2 cases of atrophy of the testicle. In any long, thorough follow-up, I am inclined to think that with any type of operation, a rare case of atrophy of the testicle will be found. There were 56 direct hernias with one recurrence, and that was in a case in which we had resected a hydrocele at the same time and atrophy of the testicle followed. So altogether there were 149 cases with 2 recurrences (1.34 per cent). In 34 bilateral cases there were no recurrences, which would seem to bear out the belief that in a good risk patient, there is no contraindication to performing simultaneous bilateral operations.

TABLE 10
FIVE-YEAR STUDY OF COOPER'S LIGAMENT REPAIR

	NUMBER	RECURRENCE		COMPLICATIONS
		Number	*Per Cent*	
Indirect Sliding (14)	90	1		1*
Direct	56	1		2**
Recurrent	3			
TOTAL	149	2	1.34	3 (2 per cent)
Bilateral	34	0		

*Atrophy of testicle.
**Hydrocele—resection.

In conclusion, it has seemed to us as a result of this study that if proper technic is used, sharp dissection, nonabsorbable sutures, careful avoidance of tension, gentle handling of tissues, by the rectus sheath, rectus muscle type of operation, preferably using Cooper's ligament in the closure, a satisfactory low recurrence rate can be obtained.

Finally, the whole question of operations for hernia, as well as other types of surgical procedures may be considered in the light of the good old aphorism of Michelangelo, which runs something like this: Trifles make perfection, but perfection is not a trifle.

Diagnostic Clinic

Abnormalities of Infants Resulting from Non-Genetic Factors

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This ponderous topic, which I am afraid I selected myself, was not selected to give the idea that I do not believe in heredity, but to emphasize that there are causes other than heredity for some congenital defects.

HEREDITARY DEFORMITIES

I should like to point out that even defects that are definitely hereditary are not necessarily the result of a defective gene specific for the affected part. There is reason to believe even that some fetal abnormalities on a hereditary basis are not the result of genes which are in any way defective. Such a condition is exemplified by the so-called congenital dislocation of the hip, which so frequently occurs on a hereditary pattern. This condition appears to be due to an excessive relaxation of the fetal ligaments induced by the relaxin of the mother and supplemented by that of the fetus itself. This hormone is a normal component of progesterone, a normal hormone which occurs in considerable quantities during normal pregnancy. The fetal hip in this condition is normal in formation, but it is not ossified because there has been a lack of pressure on the acetabulum and therefore an insufficient incentive for ossification. If this is the cause of congenital hip, as it appears to be, there is no defective gene concerned.

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Another type of hereditary defect might be mentioned in which there is a defective gene, but it is for another part and not for the defect itself. An example of this is supplied by the hereditary clubbed feet in mice described by Dr. H. J. Bagg of Chicago. These mice were discovered to have in utero a dominant hemorrhagic defect, and only those mouse fetuses whose feet were confined by a clot had clubbed feet; the others did not.

These examples are given to illustrate the fact that even in the presence of true heredity the mechanism for production of the deformity may not be what it at first appears. The involved gene may be one influencing one or more maternal factors which are reflected on the fetus, or a gene concerned with another part of the fetus quite as well as a gene for the defect itself.

OTHER CAUSES OF FETAL DEFECTS

Just as with heredity, other causes of fetal defects may exert their influence upon the mother and affect the fetus only as an indirect result, and, presumably, some may affect the fetus directly. However, examples of direct effect on the fetus are rarer than one would at first expect.

Diseases which will penetrate the placenta to attack the fetus in utero are confined to syphilis, typhoid fever, paratyphoid fever, smallpox, chickenpox, and malaria, according to the list by Potter and Adair of Chicago.

Perhaps for practical purposes it does not matter now what the mechanism is for the production of the deformity, but it should be important in a therapeutic way eventually.

At this time, considering that the etiologic

CHARLES C. CHAPPLE

agent may be influencing the mother, the fetus, or both, let us take up some of the apparent causes and the resulting defects. In general it may be stated that the earlier that any etiologic agent becomes active, the greater the chance of resulting deformity and the greater the deformity. Dr. George Corner, embryologist at the Carnegie Foundation, investigating normal sows after copulation, found that about 30 per cent of the impregnated ova were sufficiently injured, presumably through some minor change in the maternal environment, that they did not implant at all.

Observations on humans are somewhat limited, of course, though there are some. Drs. Hertig and Rock at Harvard, in investigating the embryos of women who had undergone hysterectomies, discovered that about 50 per cent of those impregnated ova contained fetuses or embryos which were already sufficiently malformed that they were not expected to be capable of surviving a normal menstruation. There are other defects, appearing later, which are less damaging and which allow the fetus to survive for longer and longer periods or even throughout pregnancy but which prevent extra-uterine life. Then, of course, there are still others which the baby can survive and which vary from the severe to the mild.

The causes of congenital defects which have been suspected or proved include chemicals such as alcohol used by the mother. This has been suspected for generations but has never actually been proved to cause fetal defects. Nicotine is another in the same category. Lead has a good deal more evidence behind it as a true producer of fetal deformity.

A definite factor in influencing the production of fetal deformity is the mother's age. It seems that her age has a fairly well established relationship to the incidence of mongolism and chondrodystrophy. There are many exceptions to this; nevertheless, the older the mother the more likely the appearance of mongolism and chondrodystrophy.

Irradiation, x-ray, or radium to the uterus in therapeutic amounts, in the first trimester of pregnancy, has been shown pretty clearly to give mental retardation, spina bifida, and deformities of the extremities.

Nutrition has been shown to affect the newborn of animals. Dr. Warkany of Cincinnati has raised rats throughout their entire lives on a riboflavin deficient diet. Their offspring had cleft palate, bony fusions, syndactyly, and micromelia. These rats had spent their entire lives on a deficient diet. He had another series of rats which were restricted in their intake of vitamin A. These rats had offspring with congenital defects of the heart, cataracts, and microphthalmus.

On a strictly dietary basis, there probably is nothing comparable to this; there certainly is not in the same degree, in the human.

The only real example of nutritional deficiency affecting the human offspring is iodine lack in the induction of cretinism.

Aside from the diseases I mentioned a moment ago as being definitely established as capable of penetrating the placenta and reaching the infant, there are certain other diseases which definitely affect the fetus, whether directly or not. One of these is measles. Ordinary measles in the first trimester of pregnancy quite frequently results in fetal death and subsequent abortion; it seldom if ever results in malformation. Such diseases as lymphocytic choriomeningitis and certain other of the virus diseases have been suspected of affecting the fetus. A committee of the Academy of Pediatrics is col-

lecting data as fast as it can on this subject, and this is one of the categories of disease which is suspect.

German measles, rubella, in the first trimester of pregnancy has been shown pretty clearly to produce in the offspring of infected mothers congenital heart, mental retardation, deaf-mutism, cataract, and microphthalmus. Apparently the earlier the infection, the more severe the deformity of the fetus. This, as perhaps you all know, was first noticed in Australia a few years ago but has had a great deal of follow-up work in this country, and there seems to be no question whatever concerning this disease and the resultant malformation.

This raises several questions: (1) What about therapeutic abortion, which I do not feel qualified to go into. (2) What about the possibility of prevention by infection of young girls before puberty, with the idea of creating permanent immunity. This is uncertain at best, since there is no skin test or other means of saying who is immune and who is not, but I think it should always be borne in mind that German measles gives a very uncertain immunity. It is recommended quite widely that young girls be exposed to the disease so that they will have a permanent immunity, but even when they have had the disease many do not remain permanently immune.

A much more common cause of deformity than these we have mentioned is one I would like to discuss now and to show you some photographs of examples. The mechanism of production of these deformities is intra-uterine relaxation, posture, and pressure. With this, as with hereditary disease, it is impossible to say really whether the influence is exerted by the mother on the baby or by the baby on itself, or, more likely, the mother affecting the baby and the baby affecting itself.

The story here is something like this. The fetus during the first four months of fetal life is free to move, extend its legs and arms, and swing around, but at about four and a half months the mother realizes that she is feeling life. The infant has not increased its motions but has become sufficiently large that he is now impinging on the uterine wall and the mother can feel it. In the second half of pregnancy the infant grows rapidly but the amniotic fluid does not increase in amount. According to a good many reports, it even diminishes. In any case we know that the normal infant is about 21 inches and 7½ pounds and has been normally in about 800 cc. of fluid, or less than a quart. This means that the fluid has been about enough to fill in between the baby's body and the uterine wall, and that the arms and legs for the most part were held fairly tight to the body. In other words, the infant has been in very cramped quarters and he has had to adapt himself to these cramped quarters by relaxation. The effect of this relaxation is to accommodate him comfortably, but it has some results on the infant at times.

The infant shown in Figures 1 and 2 has lateral bowing of both femora. He has an anterior bow also to one femur. The heel is narrow and the foot is in a mild valgus. An x-ray of the infant is shown in Figure 3. We saw this infant and found him a very fretful fellow with a deformity for which we could not account. On examination we found that if he were folded into the position of Figure 2—which, of course, he could not maintain without being held with Scotch tape, as shown—he stopped his whimpering and was happy. We also saw that this position, which obviously was not a new one to him, could account for all his deformities.

This finding intrigued us. We found that five times on the first afternoon we folded him up in this position and instantly he went to sleep; each time as we let him out he became uncomfortable. We began calling it the "position of comfort." After this we decided to look for "positions of comfort" as evidence of the fetal positions in other infants.

Figure 4 shows a very common "position of comfort," which has no deformity involved at all; it is perfectly normal. There is no deformity here. That is not a position, I think, that any of us would find very comfortable.

From the position shown, excessive relaxation begins to show in the joints of the long bones first. We begin to follow it into a backknee. This sort of backknee is exceedingly rare in the older child, but in infants we found 30 of them during the first three days of life in three months on a small service. If the backknee is less than 45 degrees, complete recovery without treatment occurs very shortly. Figure 5 is a photograph of such a child who had it for five days. In the more severe grade where the foot has fallen down 90 degrees, there is always accompanying posterior dislocation of the hip be-

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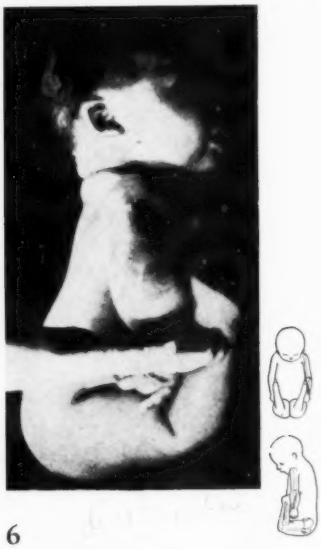

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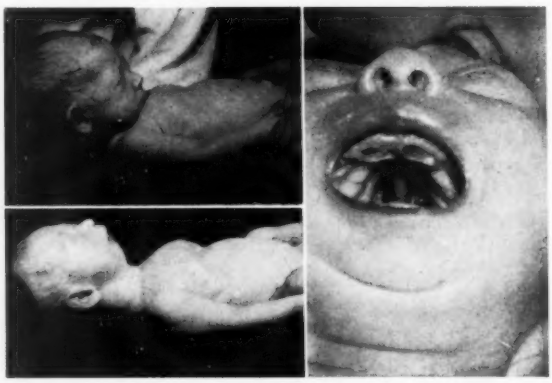

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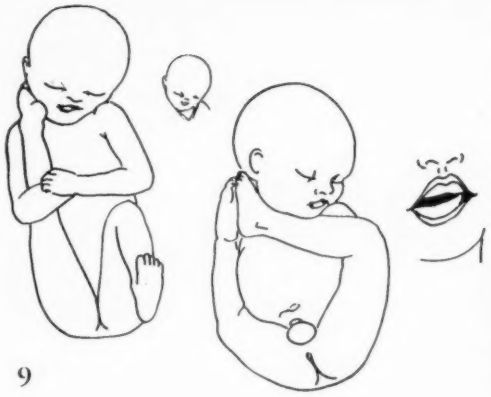

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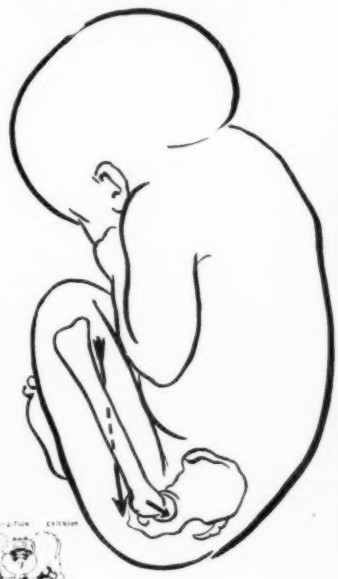

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14

cause of the uterine pressure on the knee. Frequently the elbows are dislocated because of the leverage on the foot. Figure 6 illustrates their "position of comfort."

Figure 7 shows that whether the infant's head is way up in the fundus or is on the semi-transverse line, the baby is quite able to swing all the way around in utero without being able to change its position in relation to itself. I have a good many of these and it seems to be true in all of them.

The child seen in Figure 8 was held with his head flexed beyond the normal position. He has an "Andy-Gumpism." He has almost no chin whatever. Apparently because his chin was pressed up inside his mandible and there was not room in his mouth for his tongue to develop, it developed inside his nose. We have 4 of these cases. This is a syndrome, I suppose, since these deformities are noted as occurring concomitantly, always.

Figure 9 shows a child whose leg is holding his head over on the shoulder and the chin is being forced away from that shoulder. This results in the type of jaw shown on the right. This is a very common thing. Infants held in this position sometimes have torticollis as well, from elongation of the sternocleidomastoid muscle on this side.

A photograph of such a chin is shown in Figure 10. These usually require no treatment. They may persist in a mild form but most of them correct themselves in a few months.

Positions affecting feet include those with either heels or toes forward against the head.

Normal relaxation with the foot flexed so it comes up lateral to the thigh usually results when the baby sits on its feet; relaxation of the knee occurs to accommodate that position. The resulting foot is a mild varus, but is not of any real degree and is very common.

The more severe ones (Figure 11) of this sort are from pressure not of bone against soft parts, as buttocks, but against another bone. Figure 12 shows the prenatal x-ray in which we were able to verify that.

The milder of the deformities we have seen correct themselves. In the hip the situation is different (Figure 13). In any normal fetal position the muscle pull into the hip is not great. Most of the responsibility for seating the femoral head in the socket lies on the capsule itself. The muscle of the thigh tends to pull the hip out and in the presence of relaxation of the capsule, the counter-pull of the muscle limits the pressure into the socket.

When the hip has not been pulled in sufficiently to induce the ossification of bone from the pressure stimulus, the hip remains as cartilage. Figure 14 is an x-ray of a newborn infant with such a cartilaginous hip. The acetabulum on the abnormal side is here in cartilage, but it does not show in the x-ray. Instead, you can see that there is no bony protuberance which would prevent the femur's rising on the ilium, but notice that there is very little if any dislocation or subluxation. On the normal side the femur is pointed directly in, the acetabulum is hard, and there is a fairly sharp outer margin. On the soft side there is not and the femoral head, as the muscle develops and weight-bearing begins, will be forced on up the ilium and present the condition called congenital dislocation of the hip, which is not a dislocation at birth.

Another example, this one of bilateral hips, is shown in Figure 15a. Here you can see that there is some lateral displacement. For comparison the normal state is shown in Figure 15b. Both of these pictures were taken at birth.

So-called congenital hip is recognized by the limitation of abduction (Figure 16). Using the vulva or median raphe as a perpendicular, the flexed legs of a normal baby can be abducted almost to the table. If they cannot be abducted more than 45 degrees, as in this infant, it is worth getting an x-ray of the pelvis to see whether or not the child has this persistence of the fetal cartilage.

In closing, I just want to say that recognition of this condition in early infancy makes it possible to treat most infants simply by spreading the legs in abduction with the diaper (Figure 17). The prognosis is excellent if treatment is begun in the first month—by that I mean that the baby will walk at the normal time without a limp. If recognition is delayed four or five months, physical therapy will be necessary to achieve abduction and reduction may then be required.

Figures 1-5, 9 and 13 are reproduced from "A Study of the Relationship Between Fetal Position and Certain Congenital Deformities," by Charles C. Chapple and Douglas T. Davidson, Journal of Pediatrics, April, 1941. Figures 7, 8, 14 and 17 are reproduced from Mitchell-Nelson's "Textbook of Pediatrics," published by W. B. Saunders.

15a

15b

16

17

Primary Gastrectomy for Perforated Peptic Ulcer

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Perforated peptic ulcer is one of the commonest emergency abdominal conditions, ranking second to acute appendicitis among patients requiring surgery in the Central Emergency Service of the Civil Hospitals of Lisbon (Banco do Hospital de S. José). It is the acute complication of peptic ulcer that requires emergency surgery most often. From 5 to 14 per cent of all peptic ulcers perforate at one time or another, according to Puig Sureda, Werbel, Kozoll, and Meyer. It accounted for 25 per cent of the patients with duodenal ulcer who were operated on at the Massachusetts General Hospital (Allen and Welch).

The frequency of perforated peptic ulcer seems to have increased and appears to be induced by periods of stress and worry. It is practically unknown in the first and rare in the second decade of life; it reaches its peak of incidence in the third and fourth decades, after which it decreases again, but is sometimes seen even in very old people. It occurs much more often in males than females, the ratio being about 10 to 1. Perforated duodenal ulcers are seen about 10 times as frequently as perforated gastric ulcers; however, gastric ulcer perforations increase with age.

The diagnosis of perforated peptic ulcer, with diffuse soiling of the peritoneal cavity, generally is easy if we evaluate the history and the classic clinical signs which are present in sufficient number in practically every case.

METHODS OF TREATMENT

Although a few cases of perforated peptic ulcer with diffuse peritonitis can be cured spontaneously because the omentum, the neighboring organs, and fibrin wall off the perforation, the generally adopted policy in this condition has been immediate surgery.

I have seen a few sealed perforations which I have treated conservatively with good results, like Birks and others. However, if the picture is one of perforation in free peritoneum we operate as soon as the patient's condition permits. In the majority of cases, it is possible to operate in less than one hour, with relatively little risk, but a few come to the emergency service and die in a few hours without surgery and despite intensive supportive and antibiotic treatment.

NONOPERATIVE TREATMENT

The nonoperative treatment of this condition was first employed in England in 1945 by Bedford-Turner and later by Visick and Taylor. It has given such good results in the hands of Seeley, Hogan, Henry, and Bertram (34 consecutive cases without deaths), that I believe it should be followed with much hope and enthusiasm, taking into consideration their reservations and always being prepared to operate if necessary. Briefly their method is as follows:

1. Summary examination of the patient and x-ray study; films taken in upright position, centering the diaphragm and in left lateral decubitus, centering the umbilicus.

2. Careful history and physical examination.

3. Emergency analyses: Urine, hemoglobin, white and red blood counts, differential, albumin-globulin ratio, blood chlorides, hematocrits, specific gravity of blood and plasma, plasma proteins. Repeat analyses to evaluate progress of treatment.

4. Blood amylase, electrocardiogram, x-ray of thorax, and others when necessary.

5. Relation of intake and output of fluids.

6. Continuous gastric suction with a thick Levine tube (suction should be frequently watched to be sure of its good functioning—it is the most important part of the treatment).

7. 100,000 units of penicillin intramuscularly every four hours.

8. Begin with 2 gm. and follow by 1 gm. of sulfadiazine sodic intravenously every four hours.

9. 3,000 cc. of fluids intravenously in the first twenty-four hours (including 1,000 cc. of 5 per cent glucose in saline and 1,000 cc. of 1/6 molar solution of sodium lactate).

10. Sodium chloride, blood, plasma, vitamins, and liver extract, according to results of the analyses.

11. Morphine sulfate 1.5 centigrams in the beginning and continued as necessary; generally 2 or 3 doses suffice.
12. Nothing by mouth.

From my observations, I believe this method may cure a good many patients. However, it should be employed only in hospitals with the best equipment and most skilled personnel, because it demands such perfect medical, laboratory, and nursing attention. Due to the present shortage of nurses in most hospitals, I do not believe this regimen can be generally used. Therefore, it seems to me that there is still a reason to discuss the surgical treatment of perforated peptic ulcer. In any event, it will always have a place, at least for cases in which nonsurgical therapy is unsuccessful.

ANTONIO E. MENDES FERREIRA

SIMPLE SUTURE

The old sayings of many authorities, still supported by some, that perforation is often the cure of the ulcer and the general principle of emergency surgery that in those severe conditions the objective is to save the life of the patient, have limited most surgeons to suturing the perforation by one method or another. Some add gastrojejunostomy or pyloroplasty when duodenal stenosis exists or may be anticipated on closure of the perforation. It is still the adopted procedure in most countries, especially the United States and England. I have found only three American authors who agree with the gastrectomy method in certain cases (Balfour, Bisgard, and Graves).

The fact that many more patients with duodenal ulcer are now treated medically (85 per cent at the Mayo Clinic), also accounts for the limitation of primary gastric resection in treatment of perforated peptic ulcer.

My experience [in Portugal] has shown that in patients with chronic peptic ulcer, we have to perform subtotal gastric resection (especially if the ulcer is duodenal), even in uncomplicated cases if we want to make them comfortable and able to carry on their jobs and enjoy life. For the same reasons, if we can treat perforated peptic ulcers radically, with about the same mortality and less morbidity than with palliative operations, we do the best thing for the patient and for the economy of the hospital and the country. I dare say this is true even in England and the United States, because most statistics on the immediate and late results of simple suture for perforated peptic ulcer are not at all gratifying (DeBakey, Baritell, and others).

TABLE 1

Late Statistics on Results of Simple Suture in Treatment of Perforated Peptic Ulcer

Author and Date of Publication	Number of Cases	Cases Followed	Operative Mortality, Per Cent	Postoperative Complications, Per Cent	Recurrence of Symptoms, Per Cent	Re-perforations, Per Cent
Olsen & Norgore (1946)	128		21			
Forty (1946)	100	70	17	57*	54	
Illingworth, et al. (1946)	880	666	17		70	
Werbel, Kozoll, & Meyer (1947)	574		24	41.6		17
Kingsbury & Schilling (1947)	224		12.6	50	50	6.2
Trinca (1947)	568		15.47	24.5		
Baritell (1947)	88		1.1	22.7		
Elhinney, et al. (1948)	336 (last 12 years)		10.7 (last 3 years)			
Moore & Hendricks (1948)	101		18		66.66	

*9 deaths.

TABLE 2

DATA ON THE 6 PATIENTS ON WHOM SIMPLE SUTURE WAS PERFORMED

CASE AND YEAR	SEX AND AGE	PROFESSION	HOURS OF PERFORATION	DURATION OF HISTORY	LOCATION OF ULCER	ANESTHESIA	OPERATIVE TECHNIC	COMPLICATIONS	DAYS OF HOSPITALIZATION	RESULTS	REMARKS
1 (1944)	M 54	Peasant	28	10 years	Duodenum second portion	Local and ether	Suture and epiplo-plasty	Duodenal stenosis prior to operation	53	Cured	Anterior gastro-enterostomy 37 days after first operation
2 (1944)	M 47	Peasant	36	4 years	Duodenum	Ether	Abdominal exploration	Died during exploration		Dead	
3 (1946)	M 65	Peasant	13	6-8 years	Stomach	Ether	Suture and epiplo-plasty	Postoperative cachexia	37	?	Dismissed at request, probably carcinoma
4 (1947)	M 65	Peasant	28	none	Duodenum	Spinal and local	Suture and epiplo-plasty		13	Cured	
5 (1948)	M 38	Shopseller	38	none	Stomach	Local	Suture and epiplo-plasty	Ileus	23	Cured	Gastrectomy 2 months later
6 (1948)	M 34	Postoffice deliverer	27	10 years	Duodenum	Local	Suture and epiplo-plasty	Duodenal stenosis prior to operation	9	Cured	Gastrectomy 2 months later

Statistics of well-known men and clinics published in 1946, 1947, and 1948, not included in these reviews, indicate that the results have not improved (Table 1). As will be shown, the results of primary gastric resection are on the whole much better, both for the mortality and morbidity.

PRIMARY GASTRECTOMY

THERE are definite contraindications for primary subtotal gastric resection, but I believe it can be used with advantage in more than 70 per cent of cases. Such a radical operation—even in the most carefully selected cases—should be considered only when a hospital with the necessary equipment, a well-trained surgeon with good experience in gastric resections and emergency surgery, qualified assistants, and a good anesthetist are available.

Cases selected for gastric resection depend largely on the experience of the surgeon; good clinical judgment is essential since little is known of the patient's previous condition and resistance, and the emergency of the situation does not permit a complete and time-consuming laboratory study. Several factors have to be considered to decide the type of operation to be performed:

1. Appearance of the patient as a whole and as a result of the perforation.

2. Coexistence of other chronic or acute diseases which lower his resistance, especially of the cardiovascular, renal, and pulmonary systems.

3. Hours since perforation.

4. Age.

5. Obesity.

6. Size and localization of perforation.

7. Amount and nature of fluid and foreign material (food) soiling the peritoneal cavity.

8. Degree of congestion, pathologic condition of peritoneum and neighboring organs, and dilatation of the first loops of jejunum.

9. Anatomic disposition and pathologic modifications of the organs produced by the previously existing ulcer or ulcers.

10. Degree of improvement of patient's general condition after the quick preoperative treatment and the first few minutes of complete relief of pain by use of proper anesthesia.

Because the general operative mortality increases with the number of hours of perforation and with the age of the patient, many authors do not advise resection after twelve hours or in old patients. I believe, however, that only proper evaluation of all factors mentioned can decide the type of procedure to be adopted. Every case is different and sometimes a young man with only three or four hours of perforation, without other diseases, is a much worse risk than another over 50 years of age perforated for fifteen or twenty hours, with a larger perforation and more soiling of the peritoneal cavity. Certainly the constitution, general and local resistance of the patient, and the amount, nature, and virulence of the infecting organisms play an important role.

So far as I am concerned, the contraindications for resection are:

TABLE 3

DATA ON THE 26 CASES ON WHOM GASTRECTOMY WAS PERFORMED

CASE NUMBER AND YEAR	SEX AND AGE	PROFESSION	HOURS OF PERFORATION	DURATION OF HISTORY	LOCATION OF ULCER	ANESTHESIA	OPERATION TECHNIC	COMPLICATIONS	DAYS OF HOSPITALIZATION	RESULTS
1 (1939)	M 48	Peasant	7	8 years	Stomach	Ether	Polya-Balfour		15	Cured
2 (1939)	M 56	Mason	9	12 years	Duodenum	Ether	Polya-Balfour		17	Cured
3 (1939)	M 19	Sailor	2	2 weeks	Duodenum	Ether	Polya-Balfour		8	Cured
4 (1941)	M 38	Shopseller	5	3 years	Duodenum	Ether	Polya-Balfour		12	Cured
5 (1944)	M 39	Trader	10	1 week	Duodenum	Spinal	Polya-Balfour	Small hematoma of the wall	14	Cured
6 (1944)	M 22	Shopseller	2	1½ years	Duodenum and stomach	Ether	Polya-Balfour		17	Cured
7 (1944)	M 40	Taxidriver	4	15 years	Stomach	Ether	Polya-Balfour	Wound infection	16	Cured
8 (1945)	M 57	Shopseller	4	none	Duodenum	Ether	Hofmeister-Finsterer		10	Cured
9 (1945)	M 22	Soldier	2	2 weeks	Duodenum	Local and pentothal	Polya-Balfour		12	Cured
10 (1946)	M 52	Barber	4	10-12 weeks	Duodenum	Ether	Polya-Balfour	Small hematoma of the wall	26	Cured
11 (1946)	M 49	Cork-labor	11	20 years	Duodenum	Ether	Polya-Balfour	Bilateral bronchopneumonia		Dead
12 (1946)	M 40	Porter	5	15 years	Stomach	Ether	Hofmeister-Finsterer		18	Cured
13 (1946)	M 47	Tinker	4	6 years	Duodenum	Ether	Hofmeister-Finsterer	Volvolus of jejunum		Dead
14 (1946)	M 50	Waiter	8½	none	Duodenum	Ether	Hofmeister-Finsterer		10	Cured
15 (1946)	M 36	Nurse	5¼	5 years	Duodenum	Ether	Hofmeister-Finsterer		13	Cured
16 (1946)	M 35	Electrician	2½	3 years	Duodenum	Ether	Hofmeister-Finsterer		11	Cured
17 (1946)	M 45	Peasant	7	14 months	Stomach	Ether	Hofmeister-Finsterer		23	Cured
18 (1947)	M 31	Peasant	4	4-5 years	Stomach	Ether	Hofmeister-Finsterer		16	Cured
19 (1947)	M 26	Carpenter	24	2 weeks	Duodenum	Spinal	Hofmeister-Finsterer		9	Cured
20 (1948)	M 47	Shopseller	5	2 years	Duodenum	Ether	Hofmeister-Finsterer		9	Cured
21 (1948)	M 25	Waiter	6	1 year	Duodenum	Ether	Hofmeister-Finsterer		10	Cured
22 (1948)	M 28	Waiter	6	6 years	Duodenum	Ether	Hofmeister-Finsterer		9	Cured
23 (1948)	M 34	Stower	6	1 year	Duodenum	Ether	Hofmeister-Finsterer		11	Cured
24 (1948)	M 24	Sailor	15	none	Duodenum	Ether	Hofmeister-Finsterer		14	Cured
25 (1948)	M 43	Trader	14	8-10 years	Duodenum	Ether	Hofmeister-Finsterer	Left pleural empyema	121	Cured
26 (1948)	M 47	Peasant	7	8 years	Duodenum	Ether	Hofmeister-Finsterer		10	Cured

1. Poor general condition of the patient.

2. Coexistence of other diseases.

3. Severe spreading peritonitis with much purulent fluid and marked congestion and dilatation of the first loops of jejunum.

4. Marked obesity, anatomic disposition, pathologic modifications of the organs and localization of the perforated ulcer (in the cardiac end of the stomach and in the duodenum very far from the pylorus), which may make the operation very difficult, long, and hazardous.

I would like to emphasize that this last group of contraindications rarely are really burdens technically, except for marked obesity, because as a rule the emergency gastrectomies are easier than the selected ones. Due to the edema, liberation of the organs is usually easier and, in addition, generally there are not many adhesions—unless the patient had a previous perforation or operation on the stomach, there is another penetrating ulcer on the posterior wall of the duodenum, or another nonperforated chronic ulcer in the stomach.

REPORT OF CASES

During the nine years from November, 1939-November, 1948 I have seen 37 patients with perforated peptic ulcer. Only 4 (cases 3, 4, 24, and 25) were private patients. We divide these 37 patients into 4 groups:

(1) 2 patients who died without surgery within a few hours despite the most intensive supportive and antibiotic treatment.

(2) 3 patients, although emergency cases, who had suf-

FIGURE 1. Specimen of Case 8 showing a large hole of a perforated subacute duodenal ulcer.

FIGURE 2. Specimen of Case 8 showing the duodenal perforation and a large chronic gastric ulcer of the lesser curvature.

ficient evidence of walling off of the perforation. All 3 recovered with medical treatment and were resected from one to two months later with complete recovery.

(3) 6 patients who were not considered suitable candidates for resection. Five had simple suture and epiploplasty: 1 died of sudden collapse during abdominal exploration.

(4) 26 patients who were considered good risks for radical treatment and had partial or subtotal gastrectomy.

Groups 1 and 2 will not be considered. The main features of group 3 are summarized in Table 2. The group 4 cases (Table 3) constitute the material for this discussion.

Sex—All 26 patients were male.

Age—Average 38.5 years; youngest 19, oldest 57.

Number of hours of perforation before operation—The majority of the patients (23) were operated on in the first twelve hours, 2 were operated on between thirteen and eighteen hours, and 1 twenty-four hours after perforation. It should be noted that the 2 deaths were in patients 47 and 49 years old, both operated on before the twelfth hour (four and eleven hours).

Duration of symptoms prior to perforation—All except 3 patients had a more or less characteristic history of peptic ulcer for periods varying from one week to twenty years; in the majority (19) there was a typical history of chronic ulcer for one year or more. In 3 of the patients (8, 14, and 24) the most careful inquiry even after recovery was absolutely negative and the very first symptom of ulcer was the perforation in spite of the fact that all had pathologically chronic ulcers and in 1 (case 8) there was even seen a large perforation of a subacute duodenal ulcer and a large, definitely chronic ulcer of the lesser curvature of the stomach (Figures 1 and 2).

ONLY 3 patients had complicated ulcers (case 10, repeated previous hematemesis; case 15, duodenal stenosis and symptoms of pyloric obstruction; case 20, previous repeated hematemesis and shortly after x-ray examination another hematemesis of moderate size together with the onset of the perforation). Cases 1 and 2 perforated when hospitalized for treatment of the ulcers in other hospitals.

Diagnosis—The diagnosis was made early in 24 patients. There was a complete error in diagnosis in case 19 (ruptured appendix with diffuse peritonitis). The abdomen was opened through a McBurney incision and the true diagnosis quickly made by the characteristic nature of the fluid found and the aspect of the appendix that had only signs of superficial appendicitis. The appendix was removed, the wound closed, and subtotal gastrectomy done through an upper midline incision.

The other error was in case 24; it was only a partial error, since the question of perforated ulcer was considered in third place. Perforated subhepatic appendicitis or perforation of acute cholecystitis had been thought more probable. Gastrectomy was performed

FIGURE 3. Specimen of Case 12 showing the large hole of the perforation on the anterior wall of the stomach near the lesser curvature and a larger hole on the posterior wall corresponding to the penetration of the ulcer in the pancreas.

FIGURE 4. Specimen of Case 12 showing the ulcer of the lesser curvature and the bridge separating the two branches of the V tunnel—one opening in the anterior wall and the other penetrating into the pancreas.

through a right paramedian incision and the appendix was removed through the same incision.

General condition—This was considered good in 20 patients and fairly good in 6 (cases 10, 11, 13, 18, 19, and 25). Only 3 patients (cases 15, 17, and 18) were admitted in deep shock; all recovered before operation following less than two hours of appropriate treatment and their condition improved even more after a few minutes of complete anesthesia. More time for improving the general condition and for recovery from the collapse and toxemia was needed in some of the patients who had simple suture (it took seven hours of intensive supportive and antibiotic therapy in case 7—Table 2).

LOCATION OF THE ULCERS

Duodenum—In 22 patients the perforated ulcers were in the duodenum from a point just distal to the pylorus to about 4 cm. distal to the outlet. In case 5 the ulcer was at the jenus superior, 9 were justa pyloric (duodenal side), and 11 between 2 and 3 cm. from the pylorus.

All except 2 (cases 19 and 24) were chronic ulcers varying from ½ to 1½ cm. in diameter and all were surrounded by areas of induration of variable size. The ulcers in cases 19 and 24 were subacute.

The size of the perforation varied from 1 mm. to about 1 cm. (Figures 1 and 5).

All except 1 were freely open, though sometimes surrounded by false membranes of fibrin. Case 22 had a pin head perforation of a chronic ulcer that appeared almost sealed. The ulcers were single in 19 cases.

There were 2 cases of multiple ulcers (cases 8 and 13). Case 8 had a large perforation of a subacute ulcer of the duodenum and a large chronic ulcer of the lesser curvature of the stomach (Figures 1 and 2). Interestingly enough, this 57-year-old salesman had had no previous stomach trouble whatsoever. Following an easy subtotal gastrectomy he made an uneventful recovery. Case 13 had kissing ulcers; a 3 millimeter perforation of a chronic ulcer of the anterior wall of the duodenum and a chronic ulcer on the opposite surface. Operation was easy (the whole procedure took only fifty-five minutes), but the patient died on the ninth postoperative day with an unrecognized complication.

In many cases the duodenum was firmly adherent to the pancreas and probably there had been kissing ulcers; however, on close observation of the specimens only one (case 13) was recognized.

Stomach—The perforated ulcer was in the stomach

FIGURE 5. Showing a 1 to 2 mm. perforation of the duodenum.

in 5 patients; all proved to be histologically benign. As these 5 perforations are quite interesting, we will describe them separately:

Case 1—Perforation about 8 mm. in diameter in the lesser curvature of the stomach at the angle. Mass of induration 2 by 3 by 2 cm. Round chronic ulcer about 2.5 cm. in diameter.

Case 7—Perforation 1 cm. in diameter in the anterior wall of the stomach near the pylorus. Induration mass about 3 cm. in diameter. Chronic ulcer about 2 cm. in diameter.

Case 12—Perforation about 1 cm. in diameter in the center of a mass 4 by 3 by 3 cm. in anterior wall of the stomach at the level of the angle. The mass was adherent to the pancreas and transverse mesocolon. Observation of the specimen revealed a very interesting lesion: In the lesser curvature about 5 cm. from the pylorus there was a round crateriform chronic ulcer about 2.5 cm. in diameter. About midway in the thickness of the wall, the crater divided into a V-shape making two tunnels. One of these opened in the anterior wall of the stomach and corresponded to the perforation; the other corresponded to the part of the ulcer that penetrated the pancreas and transverse mesocolon, forming the mass (Figures 3 and 4). Between the two branches of the V there was hard, fibrous tissue about 2 cm. wide and 1 cm. thick.

Case 17—Perforation ½ cm. in diameter in the lesser curvature of the stomach, about 5 cm. above the angle. Mass 3 by 3 cm. adherent to the pancreas and the liver. Macroscopic examination of the specimen showed a large elliptic chronic ulcer (5 by 3 cm.) with the long axis perpendicular to the lesser curvature. The ulcer occupied the lesser curvature and portions of the anterior and posterior walls of the stomach. The portion of the ulcer of the lesser curvature and posterior wall penetrated in the liver and pancreas forming the mass and the portion of the anterior wall opened partially in the peritoneal cavity.

Case 18—Perforation about ½ cm. in diameter of a round chronic ulcer 2 cm. in diameter, high in the lesser curvature of the stomach, about 4 cm. below the cardia.

METHOD OF TREATMENT

Preoperative—As soon as the diagnosis was made or strongly suspected and the necessity of an emergency operation established, we instituted measures to relieve pain, combat shock and infection, and aspirate the stomach. From 1 to 2 centigrams of morphine were given subcutaneously. Saline and 5 per cent glucose solutions were given in the necessary amount to reestablish the fluid and electrolyte equilibrium by intravenous or subcutaneous routes. Transfusion of 500 cc. of blood or more if indicated, 300 mg. of vitamin C, and from 100,000 to 200,000 units of penicillin plus 1 gm. of sulfathiazole intramuscularly every four hours.

Anesthesia—All except 3 of the gastrectomized patients were operated on under general anesthesia with ether and oxygen. In cases 5 and 19 spinal anesthesia with "raquineocaine" and percaine was used and in case 9 local anesthesia with novocain plus sodium pentothal.

OPERATION

All patients except 1 were resected through an upper midline incision. In case 24 an upper right rectus incision was used.

The peritoneal cavity was thoroughly cleaned in the beginning and at the end of the procedure, by aspiration and with sponges soaked in warm saline, paying special attention to the upper quadrants, right parietocolic groove, and pelvis. After recognizing the perforation, I always explore the duodenum and the stomach because occasionally more than one perforation is present (there were no multiple perforations in our series, and no reperforations).

In all the cases, from two-thirds to three-fourths, or even four-fifths of the stomach and the necessary portion of the duodenum to include the perforated ulcer (usually between 3 and 4 cm.) were resected.

Technic—Until 1946 (in the first 13 cases) I generally used the Polya-Balfour technic employing the Hofmeister-Finsterer procedure only when the ulcer was high in the lesser curvature, to avoid angulation of the afferent loop of jejunum (cases 8 and 12). Since 1946 I have always used the Hofmeister-Finsterer technic. On two occasions, cases 8 and 12, to make the operations easier, we made retrograde gastrectomies, sectioning the stomach first. In 2 other patients (cases 10 and 15) antecolic gastroentero-

anastomoses were made for technical advantage.

In case 8 we used long loop and completed the procedure with an enteroanastomosis at the level of the Treitz angle. In case 15 the anterior anastomosis was necessary due to the anomalous transverse mesocolon which was short on the right half and completely absent on the left half of the transverse colon making this portion of the colon retroperitoneal. This disposition began at the duodenojejunal angle. We made an anterior anastomosis with short loop (15 to 20 cm.).

In all except the first 4 cases we placed intraperitoneally an emulsion of about 10 gm. of sulfathiazole in saline; in the last 9 cases we made the emulsion of sulfathiazole in 20 cc. of a saline containing from 100,000 to 200,000 units of penicillin.

The abdomen was closed without drainage in almost all cases. Only in cases 12 and 17 were small rubber dam drains left for eight days; in case 12 we cauterized the bed of the ulcer penetrated into the pancreas and in case 17 we had to resect and suture a portion of the anterior surface of the pancreas to remove the ulcer.

Intravenous fluids were given during the operations, and in practically every patient citrate blood was running as thought advisable.

Postoperative treatment—Gastric aspiration or continuous suction was rarely used and only when indicated. Intravenous and subcutaneous fluids were given in necessary amounts to have a diuresis of about 1,500 cc. in twenty-four hours (generally 1,500 cc. of saline and 1,500 cc. of 5 per cent glucose).

In the first 7 cases intramuscular prontosil in doses of 20 cc., or 1 gm. of sulfathiazole every four hours was used, whereas in the last 19 cases from 100,000 to 200,000 units of penicillin and 1 gm. of sulfathiazole every four hours, intramuscularly, were used for five to seven days as advisable. High doses of vitamin B_1, B complex and vitamin C were given daily. Nothing by mouth was allowed for the first forty-eight hours. After this the patients were given small amounts of water, and gradually began the postoperative ulcer diet reducing the fluids parenterally proportionally. Transfusions of citrated blood were given when necessary, although they were used rarely after surgery.

All patients started the ambulatory program in bed as soon as they awoke from anesthesia and were up and about twelve to forty-eight hours after surgery, depending on their condition.

POSTOPERATIVE COURSE

The postoperative course was generally very good—almost as good as in the selective gastric resection cases and definitely better than in the perforations that were simply sutured. Excluding the 2 patients who died, there was a remarkably reduced number of postoperative complications (Table 3).

Only 2 patients had serious complications: case 12 had what looked like a lower left lobar pneumonia postatelectasis but recovered and was dismissed on the eighteenth postoperative day; case 25 had an abscess of the margin of the anus on the seventh postoperative day and a left pleural streptococcus empyema starting on the tenth postoperative day. The abscess was incised, the empyema was repeatedly aspirated, penicillin was injected intrapleurally, and general supportive and antibiotic treatment was carried out for eight weeks. As the condition did not improve, external operative drainage was established and the patient recovered and was dismissed on the one hundred and twenty-first postoperative day.

Days of hospitalization—Average, 17.9 days; minimum, 8 days; maximum, 121 days. If case 25 is excluded, the average is 13.5 days. We think this is a fair average, since in the other 23 patients including the complicated cases, the average periods of hospitalization varied from 8 to 24 days and most patients were in the hospital only from 10 to 17 days despite the fact that only 4 of them were private patients.

CAUSES OF DEATH

In this series of 26 cases we lost 2 patients. It seems reasonable to describe briefly these cases in order to discuss them later and probably improve on future operative results.

Case 11 (March 25, 1946)—A 49-year-old peasant with a typical history and signs of perforated peptic ulcer for eleven hours. Twenty years of typical history of uncomplicated peptic ulcer without any previous well-planned medical treatment. General condition only fair but with no evidence of walling off of the perforation. Pulse 88; temperature 37.5° C. Blood pressure 140 systolic, 70 diastolic. No history or evidence of other diseases.

On exploration under ether anesthesia abundant seropurulent fluid was seen in the peritoneal cavity and a 2 mm. perforation of a chronic duodenal ulcer (1.5 by ½ cm.) 2 cm. from the pylorus. Operation (subtotal gastrectomy—three-fourths—Polya-Balfour) easy. Condition after operation good. In spite of appropriate intensive therapy the patient died about forty-eight hours after operation with bilateral bronchopneumonia and progressive heart failure. Bronchopneumonia confirmed at necropsy and no other abnormality or cause of death was found.

Case 13 (July 15, 1946)—A 47-year-old man with typical history and signs of perforated peptic ulcer for four hours. Fairly good history of noncomplicated, never well treated, chronic peptic ulcer for six years. General condition only fair. Pulse 108, temperature 37.6° C. Blood pressure 120 systolic, 65 diastolic.

On abdominal exploration a fair amount of seropurulent fluid was found in the peritoneal cavity. Much congestion of the peritoneum and viscera. A 3 mm. perforation on the anterior surface of the duodenum, 1 cm. from the pylorus, was found. Operation (resection of two-thirds of the stomach and 3 cm. of duodenum, Polya-Balfour) easy, the procedure taking only fifty-five minutes. Condition after operation good. He did very well for the first five postoperative days. Then his abdomen became progressively more tympanitic and he vomited

frequently. On the ninth postoperative day he died.

Necropsy revealed good condition of the sutures of the anastomosis and duodenal stump. There was a fibrinopurulent peritonitis localized in the middle and right upper quadrant of the abdomen and a volvolus of the jejunum with collapse of the ileum.

DISCUSSION AND CONCLUSIONS

Our series is too small to make any conclusions from it and we cannot compare our results of treatment by gastric resection with the results of simple suture, because like all the other surgeons who believe in the radical operation as the procedure of choice in many cases, I used the suture only in the worst cases. However, it is clear that the mortality rate of the 26 resected patients is low (7.7 per cent) and the overall mortality of the operated patients is relatively low (12.5 per cent). The operative mortality for the 6 patients who had simple suture was 33.33 per cent and this because we included the patient who died during abdominal exploration before the perforation was sutured. We also considered in this percentage the patient who left the hospital thirty-seven days after operation in very bad shape and with progressive cachexia (case 3, Table 2). This patient probably had a perforated carcinoma.

We think that at least one of the deaths in our series of gastrectomies could and should have been avoided (case 13), had the real cause of death (volvolus of the jejunum) been recognized and treated properly. Whether or not the death in case 11 could be avoided is debatable; he died with a very toxic bronchopneumonia whose causative agent was not found and at operation his general condition was only fair.

Even so, when we compare our results and the results of those surgeons who adopt the same policy as I, with the results of most of those who use routinely the simple closure, we see the overall mortality of the first (about 16.5 per cent) is lower than that of the second (about 18 per cent). In addition, the number of postoperative complications is less than with the simple suture, and the days of hospitalization are reduced. Therefore, if we select properly the cases for gastric resection, on the whole we can get less operative mortality and less morbidity with this radical procedure than using the simple suture routinely.

Against the marvelous statistics of Baritell (1 death in 88 consecutive patients treated with simple suture—1.1 per cent), I could put the statistics of Nicolas (11 cases), Carayannopoulus and Christeas (13 cases), Samain (23 cases), Usùa Mariné (41 cases), or a total of 98 gastrectomies without deaths, but that would be neither fair nor significant.

I recognize that the plan of treatment and the results of Baritell are very good but with the facilities he has to carry out his plan which includes surgery, it is certainly better to follow the policy of Seeley who treated 34 consecutive cases without deaths.

Although Conde and others have not used sulfonamides or penicillin intraperitoneally or otherwise, I believe that use of these antibiotics is one of the main factors in the reduction of mortality and number of complications in this type of surgery. Two other factors contributing to reduction of mortality and complications are the almost complete abolishment of drainage and early ambulation. I use drainage only where there is dead material to eliminate, much oozing, or when doubtful of the security of the sutures.

To summarize, I will say that, in my opinion, probably the best policy to follow in hospitals where all the necessary facilities exist, is:

(1) As soon as a correct or strongly probable diagnosis of perforated peptic ulcer is made, carry out, under the most strict medical regimen, the nonoperative treatment proposed by Seeley.

(2) After a reasonable time if there is no marked evidence of sealing off of the perforation, subject the patient to surgery.

(3) If operation is decided upon, use the best judgment and if the condition of the patient and the pathology of the case permit, do a primary subtotal gastric resection. If available surgeon and hospital facilities are good for radical surgery but the nursing is not so good, it is better to adopt the policy I have outlined.

(4) If an experienced surgeon and good hospital facilities are not available, after a few hours of good supportive and expectant treatment, use the old policy of doing the least to save the life of the patient—simple suture of the perforation.

Bibliography Furnished on Request

COMING POSTGRADUATE COURSES

University of Minnesota Medical School, Minneapolis

Continuation course in proctology, intended for physicians engaged in general practice and surgery, at the Center for Continuation Study, May 22-27. Emphasis will be on those anorectal and colonic lesions most frequently seen by practicing physicians. Course includes lectures, operative clinics, motion pictures, and seminars, with Dr. George Thiele of Kansas City as guest faculty member

Yale University School of Medicine, New Haven, Connecticut

Eighth annual session of the Yale Summer School of Alcohol Studies, at Yale University, July 8-August 4, with emphasis on the problems of alcoholism in industry and a special workshop for those engaged in personnel work. Prof. Elvin M. Jellinek, Research Associate in Applied Physiology at Yale, will direct the school.

Repair of a Third Degree Perineal Tear

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THE repair of a third degree perineal tear has always been considered a formidable procedure, in either gynecologic or obstetric surgery. Such lacerations may vary in extent from a comparatively slight tear of the rectal sphincter to extensive involvement of the rectal mucosa.

Two well-established and accepted surgical principles are herewith emphasized in dissection for repair of a third degree perineal tear: (1) careful, accurate, free, adequate dissection for layer-to-layer approximation without tension or pressure, and (2) dissection starting in normal tissue and working toward the abnormal tissue.

The author[1] has previously reported a modified method for uncomplicated perineal dissection. It is particularly applicable for dissection and repair of the rectal sphincter. The technical points which vary from the standard textbook procedure are:

1. Insertion of a large dilator (4 inches long, 1 inch in diameter) into the rectum (Figures 1-6). This facilitates tissue identification for both dissection and suturing; is a mechanical aid in dissection; acts as a mold and point for counter pressure; stretches the sphincter muscle; permits aseptic, immediate, complete identification of the rectum.

2. The tissue and muscle dissection is started high—through bilateral stab wounds at 4 and 8 o'clock, at the vaginal outlet—and carried out *from above downward,* from normal to abnormal tissue (Figures 1-5). Thus, the proper dissection plane is quickly found and easily followed. There is less scar tissue, less bleeding, and tissue is less friable. The dissection is more quickly and accurately accomplished.

3. The free, 2-inch dissection of the levator ani muscles permits restoration of the physiologic action of this longitudinal muscle group (Figure 6). Adequate dissection permits proper approximation with small suture (Watters;[5] Phaneuf[4]) with minimum tension. The proper functioning of the longitudinal muscle group is necessary for complete sphincter activity. Kennedy[3] and others have shown this also in connection with the urinary sphincter physiology.

4. The torn sphincter edges are more easily found and completely dissected when approached from, and considered part of, the levator ani group (Figure 6). The dissection is started and dependent upon proper levator ani dissection.

5. The dissected sphincter muscle edges are stretched and sutured over the large rectal dilator. This produces relaxation and maintains the continuity and anatomic structure of the sphincter muscle band. The dissection and suturing (Figure 6) of the torn sphincter then are centered about the proper previous dissection and suturing of the levator ani muscles. The suturing of the sphincter for approximation of the torn sphincter edges is accomplished after suturing of the levator ani muscles. This relieves the tension of the sphincter muscle sutures.

The following technic, based on the method previously reported,[1] is considered by the writer to be the most efficient and effective method for dissection of the levator ani group and rectal sphincter muscles for repair of a third degree perineal tear.

After anesthesia and local preparation, the special large dilator (4 inches long, 1 inch in diameter) is inserted into the rectum and fixed with towel clips (Figures 1-6).

Thirty cubic centimeters of normal saline with adrenalin (3 drops) is injected under the fascial layer, in the posterior lateral wall, 2 inches with-

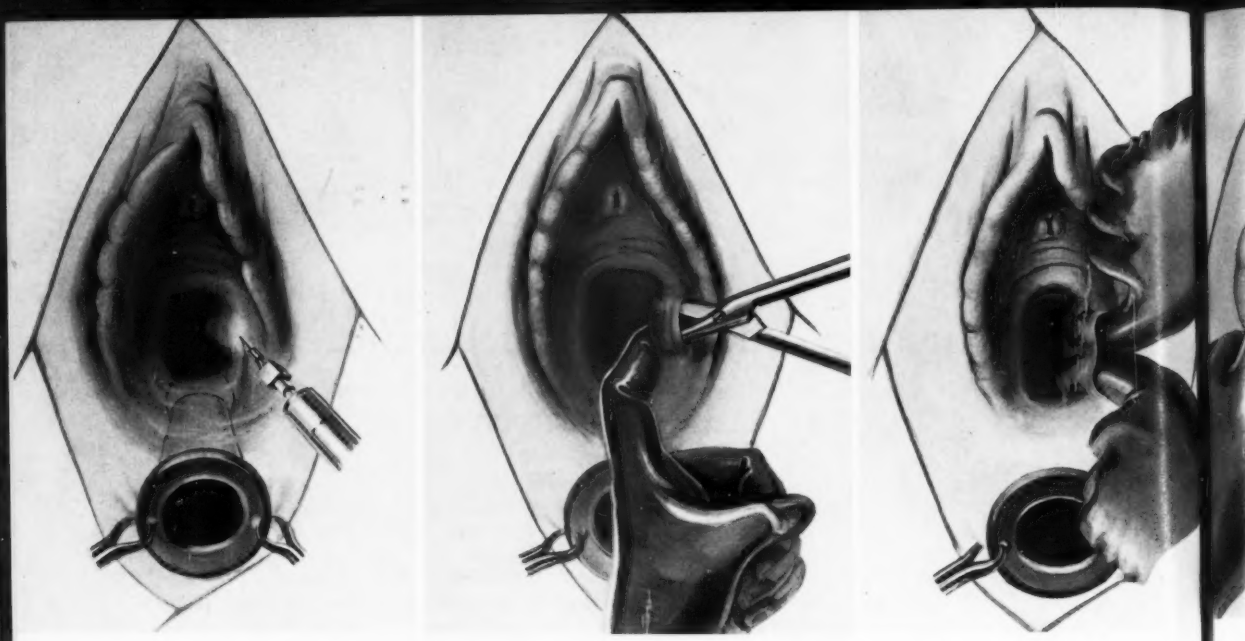

FIGURE 1. Injection of 30 cc. normal saline with 3 drops adrenalin under posterolateral wall of vagina, 2 inches within vaginal orifice, deeply about the levator ani muscle. For hemostasis and partial dissection. (To be repeated on opposite side.) An Allis forcep to catch mucosa is helpful in holding tissues and facilitating injection. A large dilator (4 inches long, 1 inch in diameter) is seen held in the rectum by towel clamps which are attached to the skin.

FIGURE 2. A 1-inch, vertical stab wound at 4 and 8 o'clock—at the vaginal orifice, made with curved, blunt-tip scissors, which are then inserted through stab wound deeply (2 inches), opened wide, and withdrawn. Proper layer easily identified by shiny, bluish-white appearance of fascia. With the finger in vagina (to guide scissors), the thickness of mucosa-fascia layer gives the feel of about 6 sheets of ordinary paper. Keep tip of scissors up, away from rectum.

FIGURE 3. Dissection of levator ani muscles with index fingers through stab wound. First one finger, later two, are inserted high up—past the levator ani muscle band. Pressure is made laterally in dissecting the muscle. Then the fingers are turned medially and structures are separated *from above downward,* away from the undersurface of the mucosa-fascia layer.

in the vaginal outlet. This is over the levator ani muscles on each side. It is for hemostasis and partial dissection (Figure 1).

Bilateral, 1-inch stab wounds are made at 4 and 8 o'clock at the vaginal outlet (Figure 2). Curved blunt scissors (held parallel to the rectum), guided by a finger in the vagina, are plunged 2 inches along the posterior lateral wall of the vagina, under the fascial layer (felt to be about the thickness of 6 sheets of ordinary paper). The scissors are opened wide and withdrawn.

The index fingers—first one, then two—are then inserted into the wound 2 inches, up past the levator ani band. This muscle is dissected through these stab wounds by lateral pressure (Figure 3).

The vaginal mucosa layer is then dissected *from above downward* (Figure 4). This method of dissection is easier because (1) this is the proper dissection layer (at the levator ani muscles); (2) there is less scar tissue; (3) the tissue is less friable; (4) there is less bleeding; (5) the dissection is from normal, toward abnormal tissue, and (6) the rectal dilator acts as a guide and a point for counter pressure in dissection.

The levator ani muscles, including the upper borders, are to be dissected for 2 inches; the rectum and torn sphincter clearly outlined by blunt dissection *from above downward.*

The posterior ends of the bilateral stab wounds are connected, allowing the usual adequate rectal flap (Figure 5). (Like the initial incision for the conventional textbook third degree tear.) The deep dissection has been previously accomplished. Completing the dissection from a known point to a known point (wound edge to dissected deep levator ani) is accomplished more rapidly (Figure 6).

THE discussion of this technic is centered about (1) a method which facilitates the adequate, free dissection of the levator ani and

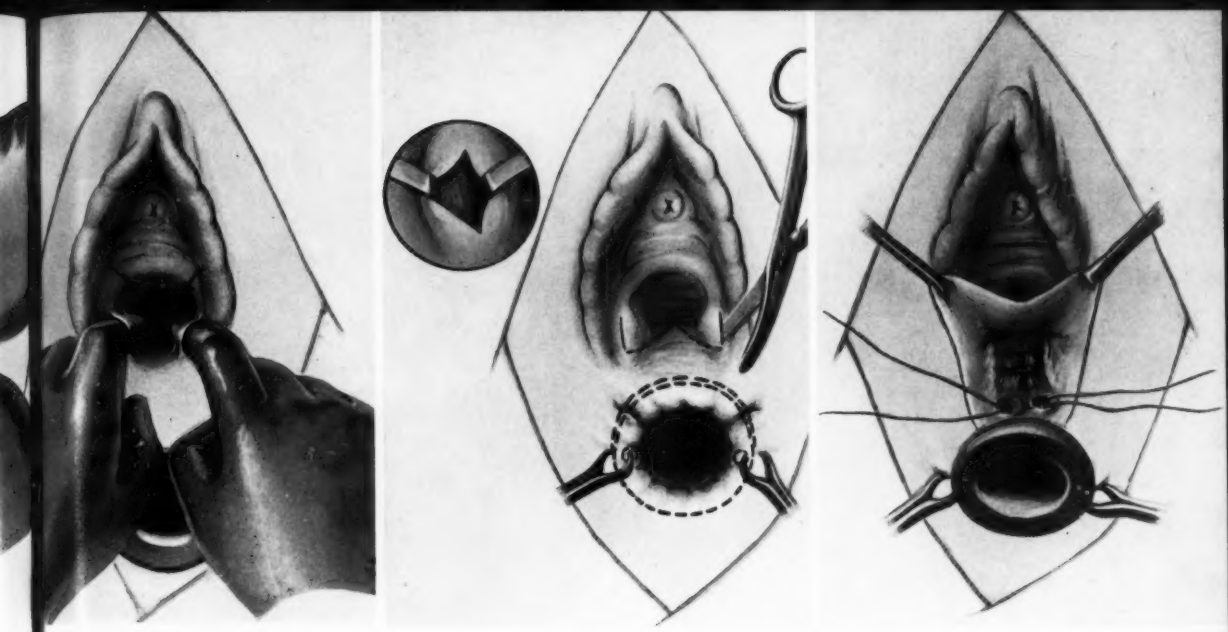

FIGURE 4. The index fingers in the left and right stab wounds, dissecting the structures *from above downward,* as much as can be accomplished by gentle, blunt dissection.

FIGURE 5. The posterior ends of the bilateral, deeply and completely dissected stab wounds are being connected, allowing adequate flap of mucosa for reconstruction of rectal lumen. *Insert:* Retracted stab wound showing muscles exposed, with chief parts of dissection quickly completed through stab wound in less vascular, non- (or slightly) traumatized region, away from adherent areas. Wound is dried with gauze and then packed with gauze dipped in saline-with-adrenalin solution. Thus bleeding is further controlled.

FIGURE 6. The dissection has been completed, with vaginal mucosa-fascia layer freed, the levator ani muscles exposed for 2 inches, the ends of the torn sphincter muscle exposed, dissected and stretched. The dissection and suturing of the sphincter are planned, realizing that it is a part of the levator ani muscle group, and that proper suturing of the levator ani should be done first to facilitate adequate approximation and functioning.

Wound closure (not illustrated): Suture—small chromic catgut (0 or 00). Interrupted for Colles fascia; continuous for vaginal; subcutaneous for skin.

sphincter edges (Figures 1-5), and (2) suturing of the levator ani muscles as the first step in approximation of the sphincter ends (Figure 6).

SUTURING of the rectal mucosa, Colles fascia, and vaginal muscosa should be meticulously carried out, as in the standard technic. Small chromic catgut (0 or 00) is advised. This discussion has nothing to offer which differs from the standard suture technic, except for the muscles. The rectal dilator may or may not be removed while suturing the rectal mucosa—and replaced for the muscle suturing. Retaining the dilator from the time the sphincter ends are sutured to the completion of the operation gives additional relaxation and stretching to the retracted, tense, and spastic rectal sphincter.

The follow-up, postoperative care remains the same: low residual diet, avoiding bowel movements for four or five days, mineral oil orally and/or by rectal injection.

The writer[2] has long been convinced that the sitz bath method for postoperative care is the method of choice in all types of gynecologic and obstetric perineal work. It has been found especially applicable in the repair of third degree perineal tears.

Six third degree perineal tears have been repaired by this method with good results.

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Diagnostic Clinic

Management of Massive Gastrointestinal Hemorrhage

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First I would like to define the type of case under discussion. In massive gastrointestinal hemorrhage I am referring only to those cases in which the hemorrhage either comes from the esophagus, the stomach, the duodenum, or the small intestine. Likewise, I am including patients in whom the blood loss has been so great that they are in an extreme degree of shock with their hemoglobin under 5 gm. and their red cells under 2,000,000, and in many instances with a blood pressure which is not obtainable. Likewise, I am excluding so-called medical hemorrhages.

Massive hemorrhages of the upper gastrointestinal tract due to ulcers of the stomach, duodenum, or gastrojejunal ulcers, in our experience constitute 65 per cent of the total massive hemorrhages seen. The other 35 per cent are due to many causes: esophageal varices, peptic ulcers of the esophagus, carcinoma of the stomach, jejunum, or duodenum, and various benign lesions of the small intestine.

Obviously, the most difficult problem in dealing with massive hemorrhage is to make a diagnosis. Many of these patients are brought in in an ambulance, they are not conscious, and a history is not obtainable; therefore, one must rely, if possible, on information obtained from relatives or friends, and to clarify this point I think a lot of confusion exists in whether one is referring to an institution with an ambulance service or to a hospital with ambulatory service.

The history is by far the greatest aid in making the diagnosis. If the patient is unconscious, it is perfectly simple to pass a stomach tube, and if the hemorrhage is coming from the stomach per se or the esophagus, the stomach is usually filled with blood; if it is coming from the duodenum or other portions of the small intestinal tract there is some blood in the stomach, but it is not filled with blood as from the other lesions. This is of real value in eliminating esophageal varices as a cause of the hemorrhage.

Obviously, a gastric analysis to determine the free hydrochloric acid in the stomach content gives one an additional lead.

Lastly, if the patient can swallow barium it is perfectly feasible to take an immediate x-ray, not so much to demonstrate an ulcer in the stomach or duodenum as to exclude esophageal varices, which can usually be done satisfactorily.

After the diagnosis has been made, the treatment of these cases always constitutes a difficult clinical problem. In our experience in the last twenty-one years we have had 325 patients with massive hemorrhage due to an ulcer, and later in this discussion I will cite the percentage operated upon and the mortality in different periods.

The means of determining whether a patient should be operated upon is merely by blood replacement. Many of these patients have the gastroduodenal, more commonly superior pancreaticoduodenal, or occasionally the right gastric artery eroded through. If blood is replaced and these patients receive 1,000, 2,000, or 3,000 cc. of blood and do not come out of their shock,

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Presented October 31, 1949, before the meeting of the Interstate Postgraduate Medical Association of North America, Philadelphia, Pennsylvania.

one has pretty conclusive evidence that one is dealing with a real arterial bleeder, and in those cases surgery must be done without delay.

It is stated in some of the recent literature that surgery is primarily a life-saving procedure. Our experience is different from that. The one danger is that many patients may be operated upon who do not need an operation, unless one is most guarded as to the real indications, as a life-saving procedure. In our total number of cases, only about 10 per cent need surgery as a life-saving procedure. The other 90 per cent can be restored to a normal state either by being operated upon as an elective procedure while the patient is still in the hospital or referred for continuous medical management if so indicated.

The one real advance in selecting these cases has been the intra-arterial transfusion, which is a very rapid means of restoring the patient to normal, and in the last year we have used that method in the extreme cases when they were brought in with no obtainable blood pressure and obviously in critical condition.

Sixty-five per cent of the lesions that we have encountered in our group of cases were ulcers; in the other 35 per cent, esophageal varices constituted half of the lesions not due to an ulcer. In other words, esophageal varices constituted around 16 to 17 per cent of the total number of cases encountered.

During the period from 1928 to 1937 the number of deaths from conservative treatment was 16, or 12 per cent mortality. I might add that half of those deaths in the early series in the late twenties and early thirties had practically no blood replacement; only the other half had some blood replacement because blood then was not as frequently used as it is at present.

The first operation I ever performed to save a patient's life was done in March, 1937. It was a simple decision as the patient was 27 years of age and had had a chronic intractable ulcer for ten years with positive x-rays. He had been transferred from the medical to the surgical service and was to be operated upon the next day, when he had a hemorrhage which we could not control by transfusions of 1,500 cc. of blood. At operation, we found that the superior pancreaticoduodenal artery had eroded through, and this patient survived.

The next 3 patients I operated were all in the hospital as cases of chronic ulcers and were ready to be operated upon the next day. One of them had 3,000 cc. of blood replacement in four hours' time; another patient had 5,000 cc. of blood in eight hours' time, with a subtotal resection during that period; during all of that time no blood pressure could be recorded. He represents the type of case to which we are referring.

There were 14 cases operated upon between 1937 and 1945 with 5 deaths—35 per cent mortality. There were 24 cases operated upon as elective operations, and here is where considerable confusion exists in the literature; you cannot tell whether many of these patients are operated upon as elective procedures or as life-saving procedures. The mortality is obviously different in the two groups.

The interesting thing is that 73 per cent of the total group were not operated upon as elective or as emergency procedures.

Before going further, I will give you the figures from 1946 to date on the same service at Bellevue Hospital. There have been 48 moderate hemorrhages; 18 massive hemorrhages with 9 deaths from conservative management; 3 operations with 2 instances in which we saved the lives of the patients. Why this should occur is difficult to explain, but from 1946 on there was a tremendous number of residents being discharged from the service and the medical service carried many more cases than normally. The residents from their varied experiences felt that blood replacement was all we needed to do in any massive hemorrhage regardless of the etiology. The tragedy was that there was a 19 per cent mortality, the highest mortality we have had in all the cases reviewed since 1928 and, incidentally, we got only 3 patients for surgery during that period of time.

I might add that this has since been corrected, and all massive hemorrhages, whether they are admitted to medicine or surgery, are immediately seen by surgeons and the indications for operation decided, but this does not mean that they will necessarily be operated upon. We will operate upon probably 10 per cent of them as emergency procedures. The others will be treated as elective surgical cases or referred for medical management.

In all the patients upon whom I have operated, including private patients, all of the duodenal ulcers have had their superior pancreaticoduodenal artery eroded through with a marked chronic pancreatitis, and, of course, the chronic pancreatitis keeps the artery from contracting

if it could. Many of our patients have been young, under 50 years of age.

Intra-arterial transfusion has been of great help in these cases. We are not using it, incidentally, in all patients, only in those critically ill. If you can restore their pressure to normal shortly and maintain it, it is not necessary to operate; if you have to keep replacing blood, it means you have a large arterial bleeder and the patient should have immediate surgery regardless of how critical his condition is.

I would like to say one word about esophageal bleeding. Many patients with enlarged spleens are having splenectomies when they have a portal hypertension, and these are usually young individuals who have had one or two massive hemorrhages. Someone has taken out the spleen and that patient is deprived of the only chance he has of being saved, namely, by a splenorenal shunt or a portal-cava anastomosis. If one encounters such a bleeder and he is operated upon by mistake, the patient should be closed and have someone who is competent to do blood vascular surgery (and there are many surgeons who can do it now), reoperate upon the patient at a later date. Once the spleen is removed the patient's chance of having permanent relief is gone. We have had 2 such patients referred to us in the last twelve months.

CASE REPORTS

A patient in the late thirties had a massive hemorrhage which was diagnosed as coming from the lower esophagus or stomach. When the patient was in condition, x-rays revealed an esophageal ulcer, and that patient was put on medical management.

In a case of carcinoma of the stomach the patient had a massive hemorrhage which required about 2,500 cc. of blood replacement before he could be operated upon as an elective procedure. We have had 3 patients with carcinoma of the stomach who have bled to death from ulcerating carcinoma on the lesser curvature.

An elderly woman, 73 years of age, had had two coronary attacks and had been in the hospital for some six weeks with constant bleeding. It had been assumed that she was bleeding from a diverticulum of her esophagus which had been demonstrated on roentgenologic examination. When we got her—within twenty-four hours of her discharge from the other hospital—she needed 1,000 cc. of blood replacement, and it was necessary to give 500 cc. a day to keep her in balance. After 6,000 cc. of blood and having her x-rayed again it was felt that she had a superficial ulcer. There was no blood in the gastric contents and her free hydrochloric acid was 73 units. She was operated upon and no real ulcer could be demonstrated in the duodenum, but her upper gastrointestinal tract was full of blood. Subdiaphragmatic vagotomy was done, her bleeding stopped immediately, and she died ten days later from a coronary. Autopsy showed a superficial duodenal ulcer which had healed in that period of time.

Another patient had a perforated duodenal ulcer. He had had a gastroenterostomy at the time of the closure some years before. He had had two perforating gastrojejunal ulcers, and subsequently had suffered from three massive hemorrhages. We have had three cases of gastrojejunal ulcers which have necessitated emergency operations because of erosion through one of the branches of the middle colic artery.

A man 73 years of age, was admitted not to Bellevue but to the former Post-Graduate Hospital with acute retention of urine, necessitating a cystotomy. While he was convalescing from that, with the blood urea in the forties, he had a massive hemorrhage. He had had a duodenal ulcer for twenty-five years; x-rays revealed a penetrating duodenal ulcer. This man had 6,000 cc. of blood in ten days' time. He was operated upon, but we made one mistake; we did, or at least I had the resident do, a gastroenterostomy because of his critical condition, and subdiaphragmatic vagotomy. (This was three years ago.) The patient did not bleed for three days. Then he bled again and had 6,000 cc. of blood replacement in three days, but died at the end of this period with an erosion of the superior pancreaticoduodenal artery.

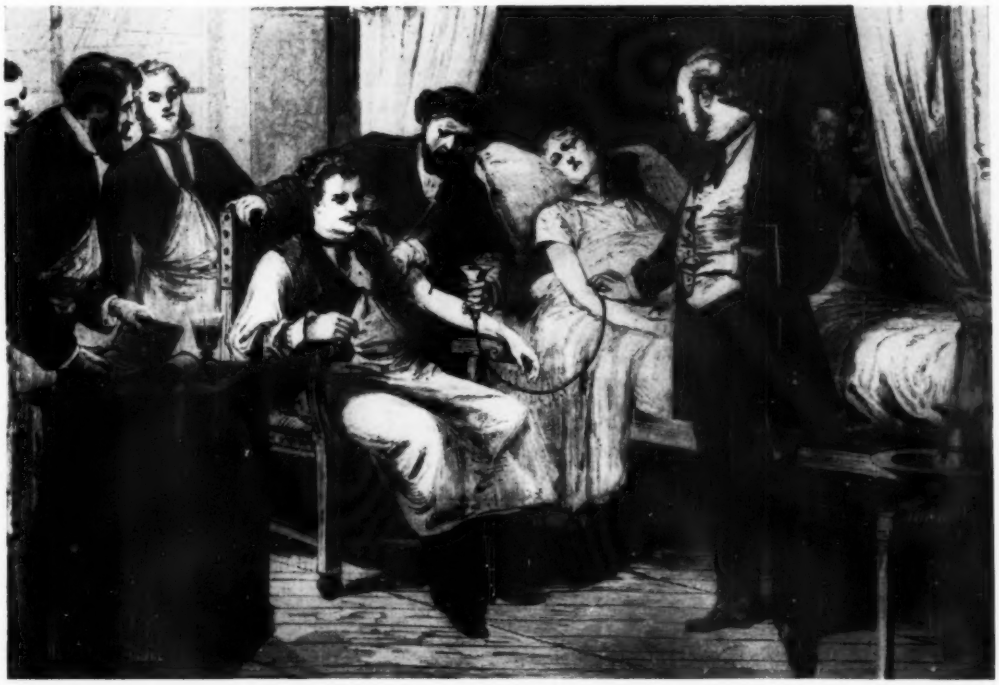
I WANT to condemn vagotomy in cases of massive hemorrhage. When one comes to surgical management in the active bleeding phase, all of these patients need a subtotal gastrectomy, as it is the one means of controlling the artery.

We had a case on the wards last week illustrating this type of case. This man was 61 years of age and lived in a camp for unemployed men in New York. He was admitted to the medical service with no obtainable blood pressure, with a hemoglobin of 2.5, red cells under a million, completely comatose, and no friends and no history obtainable. This patient had 3,000 cc. of blood replacement in twelve hours. The next day he was conscious enough so we could get a history of some abdominal discomfort for a few weeks before admission and tarry stools for one week before admission. It was decided that this patient had a bleeding duodenal ulcer and should be operated upon, but his temperature was 104° F., and x-rays revealed a bronchial pneumonia; therefore operation was postponed. In less than forty-eight hours he had another hemorrhage which necessitated 2,000 cc. of blood, and in another twenty-four hours he had another hemorrhage and his temperature then had come down to 101° F. We felt that the only

chance of saving this man's life was by immediate laparotomy, which was done with a subtotal resection. At operation the superior pancreaticoduodenal artery was found to be eroded through. His temperature is remaining at 101° F. three days after operation, his pulse is in the eighties, and now he is conscious and it looks as if the patient's life has been saved. We are convinced that it would not have been saved by any form of conservative management.

Finally, the solution to this problem (and I am now speaking of a large institution with an active ambulance service) is 100 per cent cooperation between the medical service and the surgical service. In the wards on our service there are over 100 general surgical beds and over 100 general medical beds, and they are always over-filled, which gives in excess of a 225-bed capacity. With that bed capacity we get about 12 to 14 patients per year with massive hemorrhage, a total of 325 in the last twenty-one years, that are in the category of extreme bleeders. Of that number, about 10 per cent will need surgery as an emergency procedure. Now, with the cooperation of the Department of Medicine, every patient admitted with massive hemorrhage is considered a surgical problem, and the patients needing immediate surgery can be operated upon within a few hours if they cannot be restored to a normal blood volume, or whenever it is deemed advisable in the judgment of the surgeons.

The total number of cases in the past twenty-one years that have died either with surgery or with conservative management has been 33, and the majority of those died with medical management without the opportunity of any surgical relief.



Blood Transfusion in a Hospital (About 1874)

Diagnostic Clinic

Diagnosis and Treatment of Myocardial Infarction

WILLIAM D. STROUD*

UNIVERSITY OF PENNSYLVANIA GRADUATE SCHOOL OF MEDICINE, PHILADELPHIA

As I have done so frequently, I would like to emphasize the importance of nitroglycerin in coronary artery disease, both before and after myocardial infarction. I think this drug is sadly neglected and misunderstood by some doctors and by most patients. They are afraid of it. Of course, it can be used every few minutes for pain 20 or 30 times a day, if necessary for years, and I do not feel that the average doctor gets that across to the patient.

There are some 300 active members of the Stroud coronary club. Unfortunately there are some inactive ones who didn't pay their dues regularly.

I have brought here 2 members of our club, one of the class of 1935. George, as I understand, in the fall of 1934 you were told that you possibly had a duodenal ulcer.

PATIENT: That is correct, Doctor.

DR. STROUD: But you didn't do much about it. Is that right?

PATIENT: No, I did not go on an ulcer diet.

DR. STROUD: Did you stop smoking?

PATIENT: No, I did not.

DR. STROUD: Then in Atlantic City while skating in February of 1935 you developed some symptoms. What were they?

PATIENT: A feeling as though I had swallowed a grapefruit, to put it in a crude way—an acute discomfort but not actual pain, and I broke out into a most profuse perspiration.

DR. STROUD: And then they took you to a hotel and the doctor there gave you what?

PATIENT: Castor oil.

DR. STROUD: And the next morning you came home near Philadelphia and a doctor in his seventies saw you and suggested an electrocardiogram, is that correct?

PATIENT: That is correct.

DR. STROUD: I just mention that to show what clinical experience will do over a period of years for a physician.

Then you were in bed about six weeks and you got back to work, about how many weeks after the occurrence?

PATIENT: Oh, eight or nine weeks and I was back on full duty.

DR. STROUD: And you have had no chest pain at any time, not even down at Atlantic City?

PATIENT: No symptoms at all that I know of.

DR. STROUD: Since then you have been perfectly well, smoking as much as you want, playing golf? How often do you get tight?

PATIENT: Well, that is a relative term. I don't stay away from alcohol because of your earlier advice.

DR. STROUD: That is just what I wanted you to say. And lately you have had some more duodenal symptoms, is that right?

PATIENT: That is correct.

DR. STROUD: And you have been on a milk slush diet?

PATIENT: Last July and August I was on just such a diet.

DR. STROUD: Thanks ever so much, George.

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Presented November 2, 1949, before the meeting of the Interstate Postgraduate Medical Association of North America, Philadelphia, Pennsylvania.

Now may I have the member of the class of 1942, please? We call all the members George, just like porters on the train. I forgot to tell you the slogan of the Stroud coronary club: How mild, how mild, how mild can a coronary be!

George, what were you doing when you had your attack?

PATIENT: I was in the Army and I happened to be out doing some close order drill, but, Doc, let me go back and tell them what I was doing before that.

DR. STROUD: Yes, sir.

PATIENT: Well, I was in the Army. I got this coronary on Monday and I had been out on the town with a few of my fellow officers on Saturday night. I got in very late and felt very, very bad on Sunday; in fact I had such a hangover I think it would have shown up in an x-ray. I sort of rested all Sunday, and on Monday I went out with my troop. I felt all right physically, and I was out drilling them. I thought my breakfast hadn't agreed with me. I had a sort of heartburn or a tight feeling in my chest. You take over from there, Doctor.

DR. STROUD: Did it go to your arms?

PATIENT: I don't remember that particularly. I do remember very severe pain. We were on the apron of an airport and one of my fellow officers came over to see how I was doing (he had been out with me Saturday night), and I told him that I was feeling bum and I was going into the restaurant at the airport to get myself some bicarbonate of soda, which I did, and it didn't seem to do me any good. He sat there with me, and then I started to sweat and it really dripped off me, and he said he was going to call the doctor, which he did, and they took me to the hospital.

DR. STROUD: I think it is interesting that in both these gentlemen the profuse sweating was such a big factor, as it so often is, and also that in the first case there was no chest pain and he thought he was having indigestion, as they so frequently do; in this gentleman's case he also thought so because he took bicarbonate of soda.

You were in the hospital for a few weeks when we got you home.

PATIENT: Yes, I was there six weeks.

DR. STROUD: Then you went down to Walter Reed Hospital?

PATIENT: I went to Walter Reed for two or three weeks and they sent me home on sick leave.

WILLIAM D. STROUD

DR. STROUD: And you chopped trees?

PATIENT: Yes, I did a little mild exercise around the place, nothing very strenuous.

DR. STROUD: You stopped smoking?

PATIENT: No.

DR. STROUD: How often did you get tight?

PATIENT: Well, listen, Doc, I have made one confession here, now I'm not going to make any more.

DR. STROUD: Since that time have you had any chest pain?

PATIENT: Well, I do if I walk too fast.

DR. STROUD: After meals?

PATIENT: After meals particularly; if I hurry to the office too.

DR. STROUD: After a bad night?

PATIENT: Well, if I am tired or if I have had indigestion or if I have been up too late—I won't go any further than that.

DR. STROUD: Does nitroglycerin relieve it?

PATIENT: Yes, it does.

DR. STROUD: Do you think the fact that you were all-American end has anything to do with this?

PATIENT: I don't know.

DR. STROUD: Neither do we. Thanks very much, George.

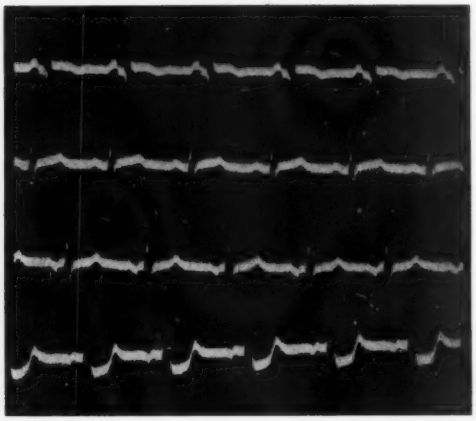

FIGURE 1. Tracing made in 1935 two days after patient had coronary occlusion while skating.

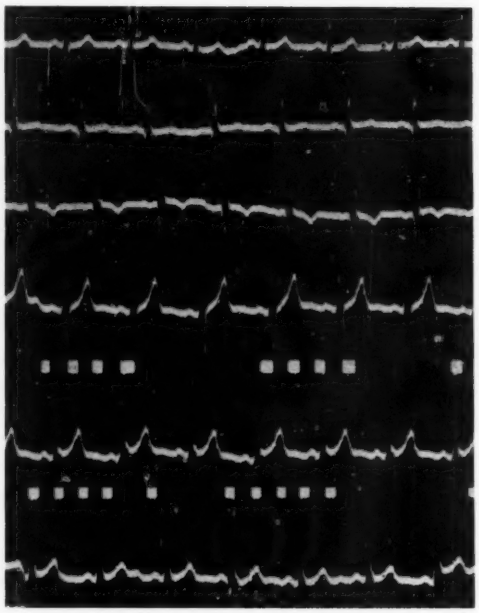

FIGURE 2. Tracing made of patient who had coronary occlusion in 1942. Figure shows that collateral circulation is developing and T-II wave which was negative is coming up.

Figure 1 is the tracing made two days after the gentleman who had his occlusion while skating at the arena in Atlantic City had his attack. That was back in 1935, fourteen years ago, and, as you see, he is now a very active individual and one of the most prominent professional men in Philadelphia.

We could not obtain the tracings that were made in Pittsburgh when the second patient had his attack but they had a negative T-II as well as a negative T-III and confirmed the diagnosis of a posterior occlusion. But you can see in Figure 2, made a few weeks later, that he is developing a collateral circulation and that the T-II is coming up.

I THINK two of the most important forms of treatment of acute myocardial infarction are rest and reassurance—reassurance by the physician, by the nurse, and by the family. Too often fear is engendered in these patients' minds. The nurse tells them they can't turn over, they can't elevate the foot of the bed, they can't listen to the radio, and, most important of all, they can't use the commode, which I feel the patient should be allowed to do no matter how severe the occlusion—that is, if he recovers in twelve hours or so. I think it is much more of a strain on the cardiovascular system to try to use the bedpan.

I feel it is very important that the doctor does not tell the patient or the family whether or not that individual is going to be totally incapacitated for many weeks and even months after the attack, because we usually do not know.

I saw a patient a number of years ago who was working in Bangor, Pennsylvania, and the job that he had was with his uncle, who could have made his job just as easy as possible. The day he had his occlusion the doctor told him he never would work again. Well, that poor man, who had 3 children and a wife, lay there two or three months thinking that he never could work. I saw him four months later. He had made a perfect recovery, and he went back not to quite as strenuous a job but to a very remunerative job, and he is going along nicely today. You cannot make up your mind about the future in these cases, and I think most of us as the years go by are astounded at how well the majority of patients with a nonfatal coronary occlusion with myocardial infarction do. If they are having coronary symptoms they can use nitroglycerin prophylactically as well as for pain, and they can usually be eased into jobs that they can carry on for many years.

As to digitalization in acute myocardial in-

farction, I feel that there is no contraindication to it if it is necessary. With respect to digitalization, doctors are very much confused at present. A great many of them were brought up on the infusion, the tincture, the whole leaf, the cat unit, and now we have the glucosides and glycosides and they are all upset about it.

A DOCTOR in the eighties in Pottstown sent us a patient at Bryn Mawr Hospital, and looking at her electrocardiograms I noted that all the S-T intervals were depressed and I thought it looked as if she had been digitalized. I called the patient's doctor on the telephone and asked him if this lady had been digitalized, and he said, "Yes, I gave her a thorough gynecologic examination."

That kind of put me back on my heels, and by the time I got across to him what I was trying to find out, he said, "She did have a little digitalis; she had a grain and a half four times a day for the last month, but," he said, "it couldn't have affected the electrocardiogram because I took her off it two days before she came down to the Bryn Mawr Hospital." These older physicians and a lot of younger physicians are very much confused about the use of digitalis. Personally I stick to the whole leaf because I don't know whether one-tenth milligram is equivalent to a grain and a half, or two-tenths milligram is equivalent to a grain and a half, and I have seen an awful lot of people overdigitalized since these glycosides came out.

As to the prognosis in these cases and what they can do, in February, 1947, a young man—I think he was 36—had a coronary occlusion with myocardial infarction in Florida, and the doctor kept him in bed six weeks, then told him he could take four steps to a chair but if he took five he would drop dead; he said the next day he could take five steps but if he took six he would drop dead. Well, he landed up here in Philadelphia in June with 3 nurses. He wouldn't let a nurse out of his sight. He had a perfectly normal electrocardiogram. It returned to normal. It was a posterior occlusion. His blood pressure was 120/70, his pulse was 72, there was no enlargement of his heart, and there were no abnormal heart sounds.

I told him to get rid of the nurses and go back to Port Allegheny where he was the president of a concern, and get to work. He said, "Can I play golf?" And I said, "Sure." So he went home and in about two weeks he kind of wondered about me and he asked the family doctor whether he could play golf, and the family doctor said, "My God, that would be suicide!"

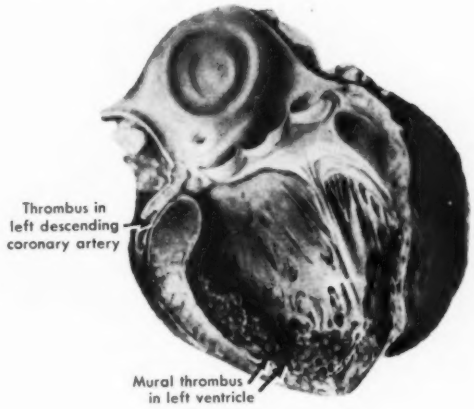

FIGURE 3. Drawing shows the characteristic thrombus lodged in the left descending coronary artery and the large mural thrombus in the left ventricle. The point of rupture of the heart is not shown here.

(From: "Coronary Thrombosis: Its Various Clinical Features," by Samuel A. Levine, M.D., with the collaboration of Charles L. Brown, M.D., Medicine, *published by Williams & Wilkins Co.)*

He was back in my office with his 3 nurses within two weeks. We decided that we had to shoot the works. He had a cottage down at Ocean City, and I sent him down there with Dr. Joseph Wagner, who is quite a guy, and told him to go to it. This was on July 15, and I got a letter from Dr. Wagner in September saying, "As you know, I first saw him on July 15, and together we engaged in a program of physical exercise (he had his coronary in February) which developed to the point that on or about August 15 he was capable of playing 18 holes of golf, swimming half a mile, walking four miles, and bicycling ten miles all in one day. Under this program he gained an appetite, his weight increased 18 pounds, and his confidence returned. He has returned to his occupation as president of a concern in Port Allegheny and it is reported to me recently that he is doing very well."

He has come in to see me every six months since 1947 and he is still working, playing golf, swimming, and so forth.

I do not advocate that as the treatment of every coronary, but it shows you, when these things heal properly three or four months afterward, that the vast majority of cases can return to useful, normal lives.

FIGURE 3, I think, illustrates beautifully just what happens in a myocardial infarction. It is a page from one of Sam Levine's articles. You get these intramural thrombi so often, and this shows you why you cannot tell the family after two or three weeks that the individual is out of danger, because there is always a possibility that he may throw an embolus or go into ventricular fibrillation. I do not mean to say that you should keep them continually frightened, but tell them that in 10 per cent of the cases something may happen suddenly.

I hope Irving Wright has gone back to New York, because I am not sold on dicumarol. I respect his opinion, I respect his work, but I do not believe—now that we use leg exercises with the patient in bed in myocardial infarction and let them out on the commode after two or three weeks, let them sit up in a chair for half an hour to an hour twice a day, increasing the time upwards—that there are as many embolic phenomena as have been suggested in the recent literature, and I am not convinced, except in cases where there are changes in the leg vessels, that dicumarol prevents embolic phenomena. It is an expensive proposition. You practically always have to take the patient to the hospital; you have to stick him for prothrombin times every day, and I just feel that we have not proved certainly that routinely every patient with myocardial infarction should be given heparin and dicumarol.

Concerning the study sponsored by The American Heart Association, in which our laboratory at the Pennsylvania Hospital took part, I feel the interpretation of the results in those 800 cases is not accurate. In 16 or 17 laboratories they took one case and did not give them dicumarol; the other case alternately they did.

As you know, what is going to happen to these patients really depends on the size of the vessel occluded, and you can have a run of large vessels occluded, very seriously ill individuals, and not give them dicumarol, and they die; you can have a run of small vessels occluded and give them dicumarol and they do not die, and the dicumarol is given credit. I ask you for a little while to think carefully before you routinely use heparin and dicumarol in acute myocardial infarction.

LISTER'S GREAT DISCOVERY

COVER PAINTING HONORS MEDICAL PIONEER

SOME eighty-five years ago, Joseph Lister (1827-1912) first used "the antiseptic system" in performing a surgical procedure. And with that operation (an amputation) the founder of antiseptic surgery initiated a new era in medical practice.

Previous to Lister's discovery that wound infection was not caused by a "miasma" in the air, but rather by omnipresent microscopic organisms which contaminated the surgeon's hands, his instruments, dressings, etc., the mortality attendant upon any surgery performed in hospitals was appalling. Basing his deductions regarding the origin of putrefaction and infection upon Pasteur's monumental studies of fermentation, Lister proved that it was possible to prevent and to cure infection in surgical wounds, and to restore to hospital surgeries their proper function as places of healing rather than sites of lurking death.

Using as his first antiseptic agent full-strength carbolic acid, Lister later found that a dilution of the acid was sufficient to effect asepsis in wounds.

In Edmund Kopietz' cover painting, Lister is shown during a surgical demonstration, emphasizing to students and observers the importance of strict asepsis in the performance of the operation. In contrast to Semmelweiss' crushing experience of having his analogous theories of the origin of puerperal fever ridiculed and disregarded, Lister's discoveries were acclaimed and accepted during his lifetime, bringing him honors and fame among his colleagues and the laity in Europe and America.

Diagnostic Clinic

CAUSES OF Unsatisfactory Results After Operation on the Gallbladder and Bile Ducts

RICHARD B. CATTELL*

THE LAHEY CLINIC, BOSTON

OPERATION on the gallbladder and the bile ducts, when performed for patients with colic due to gallstones, with or without jaundice, is followed by a very high percentage of satisfactory results.

In the presence of dyspeptic symptoms without colic, particularly in those patients without gallstones, the results are by no means satisfactory, being no better than 50 to 60 per cent.

Gallbladder operations are among the most common abdominal procedures and every surgeon who performs abdominal surgery is confronted with the problems that are concerned with this discussion.

The good results are largely dependent upon the care with which these patients are selected for operation, in order to make certain that one is operating upon a patient with symptoms which are relievable by removal of the gallbladder. When these patients are not fully studied preparatory to advising operation, we are very apt to overlook other conditions which will produce similar symptoms and which, of course, will not be relieved by removal of the gallbladder. Then, too, there are other conditions that may be found in the biliary tract that may not be sufficiently appreciated as a cause for symptoms. In other words, the gallbladder may be removed for stones and stones may be removed from the common duct, and yet within a relatively short period of time the patient will have the same symptoms that were present before, because of an overlooked condition at the lower end of the common bile duct—fibrosis of the sphincter of Oddi.

It is essential that the operative procedure answer all of the problems that may be present related to the gallbladder and the common duct. The commonest conditions that may be confused as a cause of symptoms related to the gallbladder are those related to the gastrointestinal tract as, for example, by the dyspeptic syndrome, which can be produced by diaphragmatic hernia; by a small posterior wall duodenal ulcer, or by functional conditions in the remainder of the gastrointestinal tract, such as a spastic or irritable colon.

Twenty years ago, my associate, Dr. Kiefer, and I reported before the American Medical Association on a group of cases which we considered "Failures after Cholecystectomy," and in our own experience, one-half of the failures, that is, one-half of the patients who had continuing symptoms after cholecystectomy, were due to some other lesion that had been present at the time the cholecystectomy was performed. Unless we do more of a study than that directed just to the gallbladder, in the patient suspected of having gallbladder disease, we will continue to make errors of this type. With the exclusion of the patient with acute cholecystitis, we have the opportunity and the time to do the appropriate studies to exclude these other lesions. These should include fluoroscopy of the esophagus and of the stomach and duodenum to determine whether there is a diaphragmatic hernia, cardiospasm, or ulcer present. They should include a barium enema to exclude a spastic

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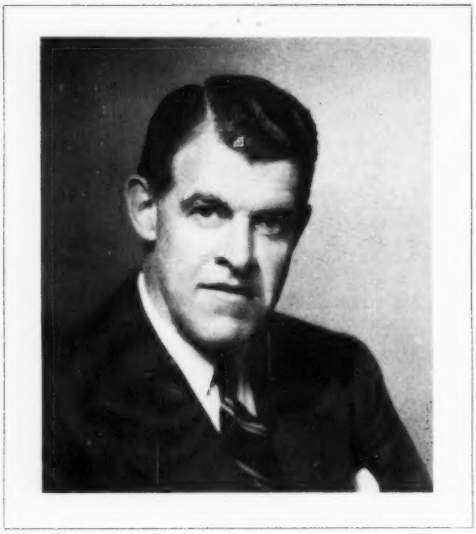

RICHARD B. CATTELL

condition of the colon. Do not forget the necessity for doing a gastric analysis because, even though we have the typical syndrome associated with biliary colic, if there is an achlorhydria and this condition is not corrected, then we may have an unsatisfactory result in spite of the relief of colic.

We should be prepared, in the event of discovery of these other lesions, to take care of them either at the same time or subsequently by appropriate therapy, usually directed by our medical colleagues in the postoperative period.

If we anticipate good results following cholecystectomy, we must exclude all patients who do not have calculous gallbladder disease. That does not mean that we do not make mistakes in diagnosis and advise operation for a patient with a nonfunctioning gallbladder as shown by the dye test; perhaps it is the best policy at the time—without finding other abdominal disease—to remove the gallbladder.

Recently, at the International Surgical Society meeting in New Orleans, Professor Demel of Vienna presented this subject. He divided the cases that were unsatisfactory after operations on the biliary tract into two groups. The first group included those in whom there was a technical error at the time of surgery, and also those in whom the surgeon did not appreciate the full pathology which was present. The second group was comprised of patients who, in spite of good diagnosis, adequate operative technic, and careful follow-up, still had persisting symptoms.

Perhaps the commonest type of technical error is trauma to, obstruction or actual removal of, a portion of the biliary ducts themselves, and I will show you examples of this.

Failure to relieve symptoms following operation when the findings seem localized to the biliary tract is sufficiently frequent to cause real concern. A young doctor's wife came to us about six months ago with typical acute cholecystitis, with associated hydrops and empyema of the gallbladder. She had a normal common duct and, as is usually the case, an impacted stone in the cystic duct was found. Cholecystectomy, with exposure but not exploration of the common duct, was carried out. In four months she returned, stating that she had had six attacks of pain subsequent to the operative procedure. Careful studies at that time failed to reveal any cause for disease, and there was no fever.

This is the most difficult problem that we as surgeons have to meet. There are few diagnostic tests to help us. Perhaps the best and most effective is the diagnostic biliary drainage. By entubing the duodenum, making certain of its position by fluoroscopy, if we aspirate the duodenal content and analyze it microscopically we may get very valuable information as to the cause of persisting symptoms. If both cholesterin crystals and bilirubin pigment are present we can be assured that a calculus is still present in the biliary tract. If we find in the sediment high columnar sheets of epithelial cells and a large number of leukocytes, we can anticipate that there is a persisting inflammation in the biliary tract due to infection, usually. Estimation of the serum bilirubin or icterus index during an attack may be helpful. If an elevation in the serum amylase can be demonstrated recurrent inflammation of the pancreas may be suspected.

THERE is a number of causes of poor results following gallbladder operations that I should like to outline.

The first is the poor result following cholecystostomy. I do not wish to imply that the operation of cholecystostomy should not be used under certain circumstances. In our clinic, however, it is limited to about 1 per cent of the cases, usually in empyema of the gallbladder in poor risk patients or after perforation of the gall-

bladder. We have a patient present today, through the courtesy of Dr. Bates and Dr. Frobesi, to illustrate this point.

This patient, Mrs. B., was 58 years of age in 1944 when she had a cholecystostomy performed for cholelithiasis. In our experience, this is followed by recurrent symptoms in a large proportion of cases. Indeed, in an article from our clinic in 1928 we reported that only 27 per cent of patients in whom cholecystostomy was performed have had absence of further symptoms. Mrs. B. had a recurrence of symptoms within a few months, so that in July, 1944, a small contracted gallbladder was found and removed. The common duct was explored. It was filled with debris and sludge-like material resembling clay. The T tube was placed in the duct and was removed in three weeks.

In October, three months later, she had recurrence of right upper quadrant pain and jaundice; she was operated upon for the third time and the common duct again contained sludge. This was removed, and she then went four years without further symptoms, when attacks of pain and transient jaundice developed. In September, 1949, approximately five years later, she was again seen in an attack, and the various studies for liver function showed beginning cirrhosis of the liver because of persisting obstruction of the biliary ducts. She was operated upon, and sludge and stone were removed. No stricture of the duct was found. Thank you, Mrs. B.

The T tube is still present in this patient, having been in place approximately six weeks.

What was not done that should have been done originally? The initial procedure of cholecystostomy need not be discussed further. We are coming to appreciate more and more that if there is any remaining obstruction in the biliary duct (and the commonest site in these cases is at the ampulla due to contraction or fibrosis of the sphincter of Oddi), steps should be taken to take care of this at the initial operative procedure. That is the first thing that can be done. The second thing is drainage of the biliary duct system for an appreciable time.

The question that has been asked me on this record is: What would you do under the circumstances? In my opinion it is not enough to explore the common bile duct and make as certain as possible that all calculi are removed, followed by drainage by catheter or T tube. One must determine whether there is fibrosis of the sphincter of Oddi. At the initial procedure one should dilate the sphincter, employing graduated probes or malleable dilators such as those described by Bâkes. If it dilates with some difficulty or if the probe is not passed through the ampulla of Vater without difficulty, one should do a transduodenal exploration of the ampulla and a division of the sphincter of Oddi under direct vision. There are very ingenious instruments available for division of the sphincter of Oddi in a blind fashion with a concealed knife by passing the instrument down through the common duct. We think it is much better to expose the ampulla and do a direct division of the sphincter where it can be seen and bleeding controlled if it occurs.

Even when this has been done and drainage of the common duct instituted, we have noted recurrence of symptoms, so it is best to put a mold through the ampulla in the form of a long-limbed T tube which we have described, that passes well down into the duodenum. This tube should be left in place in these complicated cases for a minimum of twelve months to prevent contraction of the sphincter. During this time the T tube is irrigated twice daily with 15 to 33 cc. of normal saline solution, and during this period, a cholagogue in the form of decholin is given three times a day.

With this management we should be able to avoid this distressing complication of recurrent common duct stones.

A SECOND cause, fortunately rare, of symptoms after cholecystectomy is a retained cystic duct stone. We see 2 or 3 patients a year with typical symptoms of colic without jaundice, without alteration of liver function, with all tests, including duodenal drainage, negative, with the same symptoms that the patients originally presented. Even though one of our leading surgeons in this country has stated that a considerable portion, perhaps as much as ¼ to ½ inch of the cystic duct should be left in place at the time of cholecystectomy, I believe that statement was made solely to protect the common duct. If one will carry out the cholecystectomy under direct vision, all of the cystic duct can be removed without interference with the integrity of the common and hepatic ducts, and it is only by a careful dissection of this sort that we will avoid this infrequent complication and poor result.

Womack has called attention to another con-

dition causing a poor result after cholecystectomy that I am not sure we have seen—he has encountered a neuroma of the nerve supplying the gallbladder. He has confirmed it histologically and its removal has been followed by relief of symptoms. In recurrent operations on the biliary tract we have, a number of times, taken out material which on histologic section showed nerve tissue, but I am not sure personally how often this condition, to which Womack has called attention, actually produces symptoms.

We have another example of overlooked common duct stones which is a common cause of symptoms after cholecystectomy. This is Mrs. L., who is 50 years of age and who entered the hospital originally with diabetes; her studies showed cirrhosis of the liver and gallstones. She had had severe rheumatic heart disease with episodes of congestive failure. She had a small fibrosed gallbladder, which, of course, is almost pathognomonic of pathology in the common bile duct. Two stones were removed from her common duct.

During her postoperative recovery, a cholangiogram was done, and it showed a stone in the ampullar portion of her common duct. An attempt was made, after the suggestion of Best and Pribam, to try to dissolve this stone or to get it discharged through the ampulla, but this treatment was unsuccessful, as our experience would bear out. This stone could not be removed by the surgeon by exploration through the common duct, but he was able to remove it by the transduodenal approach. This type of operation was recently described by Mahorner, but I would say from our experience that it is rarely necessary to remove a stone from the common duct by this means.

This patient had a long T tube put in, such as I have called to your attention, and left in place for three weeks. This is probably not long enough, based on the points that I have brought to your notice. If you have a sphincter which dilates easily and you determine by mechanical means, by means of stone forceps, irrigation, scoops, and so forth, that the common duct is clear, it is not necessary to use a long T tube, but a short-limbed T tube should be used. When there has been considerable trauma incident to removal of a stone impacted in the ampulla I would suggest leaving the long T tube in for at least seven months. We think it advantageous always to drain with a T tube of some sort, but under these circumstances, when the ampulla is dilated easily, it is not necessary to have prolonged drainage.

We have two examples to demonstrate to you of traumatic injury to the common duct. This, of course, is a preventable situation. As Dr. Walters, who is in the audience, has said, these cases may occur in any surgeon's experience, but they rarely occur as one's experience in biliary tract surgery increases.

Mrs. M., the third patient, was 62 years of age when she had a cholecystectomy and choledochostomy in 1943 for gallstones. The T tube was left in for ten months and she was then well for two years, following which she began to have pruritus and jaundice and was found to have a bilirubin of 10 mg. per 100 cc. Three years later, in 1946, she had severe biliary cirrhosis. This was an obstructive cirrhosis and not of the type seen in patients with infectious hepatitis. In these cases the liver will recover very satisfactorily if the obstruction does not persist too long. At operation a reconstruction of her common duct was performed. The T tube was left in for eighteen months and it was irrigated three times a week. She has now gone three and a half years without jaundice, with stools of normal color. Thank you, Mrs. M.

At her second operation, which I did not discuss in the presence of the patient, a defect approximately 2 cm. long was present. Of course, her interval of freedom from symptoms for two years was the result of the fact that a common thing occurred that occurs in all obstructed ducts of this type; there is always a spontaneous internal hepaticoduodenal fistula. We are frequently lulled into a false sense of security by the presence of these spontaneous fistulas, because the symptoms may be completely relieved for a time, but you can predict that the symptoms will recur, because these spontaneous fistulas do not remain open.

This patient had a bridging of her defect by the T tube. The ends of the common duct were not approximated, yet the patient has gone several months now without recurrence of symptoms. It will be necessary to consider further surgery for this patient at a subsequent date based on the experience of everyone with this type of operation which bridges a defect by a tube without approximation of the duct ends.

The next patient illustrates another type of repair of a traumatic injury of this sort. This

is Mrs. D., who was 50 years of age when she was admitted with diabetes mellitus and biliary obstruction and jaundice of just three days' duration, accompanied by fever. Her van den Bergh was 3 mg. per cent. She was prepared for two weeks, during which time the diabetes was controlled. She had cholelithiasis; there was a marked inflammatory reaction in the gastrohepatic omentum. Although the choledochostomy was carried out satisfactorily, a probe could not be passed through the ampulla into the duodenum. Because of the poor condition of the patient, this operation was discontinued and merely T tube drainage instituted.

That is not a bad thing to do. If you have a patient with severe biliary obstruction, with high fever, a two-stage operation is not inadvisable if drainage of it with relief of symptoms will permit repair at an optimum time of a stricture if it be present.

This patient was operated on three months later, when they found that they were unable to get through the ampulla of Vater, so they did what was described as an end-to-side-end of the common duct anastomosis to the side of the duodenum. Subsequently her cholangiograms showed actually that the lower end of the common duct was still in place and the cholangiogram showing the T tube left in above also showed that the biliary tract was intact.

It would be possible to restore this, and I believe there is much less danger of subsequent cirrhosis of the liver if this hepaticoduodenostomy were disconnected.

Dr. Walters' experience as reported before the American Surgical Association last year stated that 80 per cent of their repairs of stricture were done by this type of operation. Actually, the reverse figures are true in our experience as Dr. Lahey and I have reported several times within the last few years a method of restoring continuity of the extrahepatic biliary ducts by dissection of the lower end out of the head of the pancreas by turning over the duodenum. In the past five years continuity by direct anastomosis of duct ends after mobilization of the head of the pancreas has been performed in 80 per cent of all the strictures that have been repaired at the Lahey Clinic.

The end results following this procedure have been the best of any type of the operative procedures that have been employed for strictures.

There is another cause or group of causes of symptoms after cholecystectomy that perhaps are difficult to determine at the time of operation and may be determined only by careful preoperative or postoperative study. These are cholangitis, or inflammation within the biliary tract, hepatitis and cirrhosis, and finally pancreatitis. Acute pancreatitis is frequently associated with biliary tract disease, perhaps 30 per cent of them with some degree of obstruction at the ampulla of Vater, usually by stone. As Mulholland has noted, in approximately this proportion of patients there is a common entrance of the pancreatic duct and the common bile duct into the duodenum, so that reflux from the biliary tract into the pancreatic duct may occur, leading to pancreatitis in these cases. Prolonged drainage of the common duct may be helpful in these cases. In some few patients it may be necessary to transplant the common bile duct, as recently described by Bowers, who has had 4 patients in whom this has been done with relief of symptoms.

THERE is another group of cases, fortunately rare, producing symptoms following gallbladder operations, and those are the overlooked malignancies. Unexplained dilatation of the biliary ducts must make one think of carcinoma of the lower end to the common duct, ampulla, or head of the pancreas. We have seen a number of patients in consultation who had choledochostomy performed and continued to drain large amounts of bile through the T tube or had a persisting bile fistula after removal of the tube. Persisting obstruction of the ampulla of Vater suggests malignancy. It is for this reason that, when one cannot get through the ampulla satisfactorily and cannot dilate it easily, one must rely on transduodenal exploration of the ampulla when biopsy of the ampulla is, of course, quite feasible. This should protect us against an occasionally bad result, which would prove fatal if it were not recognized.

Another cause of symptoms after cholecystectomy is postoperative adhesions. I would like to say that no patient who has been operated upon for gallstones has persistent symptoms because of adhesions, but unfortunately that is not quite true. If we do not cover the gastrohepatic omentum with its serosa and do not close the gallbladder bed, occasionally the duodenum and pylorus of the stomach will become wedged and adherent to this denuded area, and this will produce symptoms at times.

As we all recognize, if we operate upon patients for abdominal adhesions without the presence of actual obstruction we will accomplish little, but in these cases if a free graft of omentum is interposed or the omentum is attached in this area, recurrence of these symptoms may be prevented.

Finally, we have a group of patients about which as a surgeon I know nothing. These are the cases of biliary dyskinesia. I think if I made that diagnosis I would do so overlooking some organic condition in the biliary tract or associated with it. It is up to all of us who have patients with unsatisfactory results after biliary tract operation, to prove whether organic disease does not still persist. The importance of this is that if we allow a patient to continue with infection in the biliary tract and liver or some degree of obstruction, we will have serious and dire results.

How can we continue to improve the results that so far are good following biliary tract surgery? First, we must restrict operations on the gallbladder to those patients with calculous disease. Second, with rare exceptions these patients deserve full investigation, including gastroenterologic studies, and those who have been chronically ill should have studies of liver function. Third, we must be sure that an adequate and safe operative procedure is conducted. Fourth, we must follow our patients carefully to be sure that their symptoms are relieved, because we may have the opportunity of giving them treatment for other conditions which will still give a satisfactory result if properly treated.

WORLD MEDICAL ASSOCIATION WOULD FIX STATUS OF CAPTURED DOCTORS IN WAR TIME

To obviate the recurrence of certain hardships and limitations suffered in previous wars by physicians of the Armed Services who fell into enemy hands, The World Medical Association reports that it is laying plans to have all nations fix a status for the captured doctor which, while entitling him to the formal rights of a prisoner of war, will not actually designate him as one. This announcement was made by Dr. Louis H. Bauer, Secretary General of the Association at its world headquarters in the New York Academy of Medicine.

Although not a member of the Council (the Association's administrative unit of thirteen members), Dr. Bauer was present at the Council's annual meeting held in Copenhagen, Denmark. Officially representing the United States in the Council were World Medical Association's president-elect, Dr. E. L. Henderson, who is also president-elect of the American Medical Association, and Dr. R. L. Sensenich, immediate past president of the A.M.A. Dr. Morris Fishbein, editor of the World Medical Association Bulletin, and Dr. Bauer's executive assistant, Miss Margaret L. Natwick, also attended the meeting.

Efforts also were made at the Copenhagen meeting to solve a problem in anesthesia which, according to Dr. Bauer, "will have a direct bearing on many of the world's 500,000-odd medical practitioners." This involves international standardization, wherever possible, of gas cylinders and other anesthesia equipment as to color, size, and other details. Other matters discussed at the Council meeting are international nomenclature; medical security; medical education; the medical press, and industrial medicine.

Pointing out that the World Medical Association, organized in 1947, embraces in its membership the official medical societies of forty nations, Dr. Bauer cautioned against confusing his group with the World Health Organization. Although complementing each other the two units derive their support from entirely different sources and are wholly distinct in their nature and purpose. The World Medical Association, Dr. Bauer explained, represents the doctors and the national medical associations and is supported entirely from voluntary contributions and has only professional memberships.

The World Health Organization, on the other hand, is a subsidiary of the United Nations and, as such, represents the governments of the world. It is chiefly interested in Public Health and its membership is non-medical as well as medical. W.H.O. is financed exclusively by governmental appropriations.

Case Report

Familial Hemolytic Jaundice Complicated by Acute Purulent Cholecystitis

JAMES H. DEWEERD,[1] JOSEPH HYDE PRATT,[2] AND ALBERT B. HAGEDORN[3]

MAYO FOUNDATION AND MAYO CLINIC, ROCHESTER

THE first reference in the literature to the condition we ordinarily speak of as familial or congenital hemolytic jaundice, is generally credited to Murchison,[1] who, in 1885, gave a fragmentary description of several members of a family who were chronically jaundiced. Five years later Wilson[2] described another such family whose members had chronic splenic enlargement in addition to jaundice. The first reasonably accurate description, however, was made by Minkowsky,[3] who, in 1900, reported 8 cases of this disease in 3 generations of a family. He described chronic icterus, urobilinuria, and splenomegaly. At necropsy in 1 instance, he found hemosiderosis of the abdominal organs to have been present.

Chauffard,[4] in 1907, made a real contribution to the accumulation of knowledge regarding this interesting symptom complex, when he first described and reported the increased fragility of erythrocytes of patients having familial hemolytic jaundice to hypotonic salt solutions. Although the presence of microcytes was mentioned as being characteristic of the blood smears in this disease as early as 1900, it remained for Naegeli,[5] in 1931, to demonstrate that the characteristic erythrocyte in familial hemolytic jaundice was a spheroidal cell. He coined the term "globe cell" anemia.

Although today many clinicians[6,7] are of the opinion that a diagnosis of acquired hemolytic jaundice is made only because of an incomplete study of the family history, writers on the subject almost invariably mention a small group in which they consider the disease acquired because of atypical conditions which exist.

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ETIOLOGIC ASPECTS

Although theories as to the etiology of familial hemolytic jaundice are still controversial, two theories predominate. One, with adherents such as Naegeli,[5] Haden,[8] Dacie,[9] and Loutit and Mollison,[10] is that the primary defect is a hereditary defect of the red blood cell; that is, the production of small, thick, spherical, and abnormally fragile erythrocytes in an abnormal bone marrow. Loutit and Mollison supported this view in 1946 by demonstrating that in cases of familial acholuric jaundice red blood cells which were transfused survived normally, whereas red blood cells from a patient with familial acholuric jaundice transfused to a normal recipient were eliminated with undue rapidity.

The other view, supported by Doan, Curtis, and Wiseman[11] and McLaughlin and his co-workers,[7] as well as others, is that the primary development of pathologic phagocytic action in the spleen causes secondary changes in the marrow and circulating red blood cells. Immediate and permanent relief from symptoms after splenectomy in the majority of cases despite certain persistent demonstrable changes in the blood, is advanced as proof of this theory by its proponents. Hawksley and Bailey[12] chose the middle of the road in their statement that both spleen and bone marrow are primarily responsible and the spleen produces further distortion of the already abnormal red blood cells.

CLINICAL ASPECTS

The disease may lie quiescent as a latent form for a considerable time without the patient's being aware of any abnormality other than perhaps a sallow skin and mild chronic fatigue. Many pa-

tients who had never considered their symptoms particularly abnormal, suddenly found themselves in a hemolytic crisis of alarming severity.

These same patients may later be placed in the category of the chronically ill patient by reason of two or more acute exacerbations of hemolytic crises, each occurring after variable but frequently lengthy periods of somewhat mediocre health which characterizes the latent form of the disease.

Acute attacks or exacerbations tend to follow a fairly consistent pattern of sudden onset of severe pain in the epigastrium and left upper quadrant, fever, nausea, vomiting, and progressive jaundice. The spleen enlarges rapidly and is exquisitely tender to palpation. The number of erythrocytes falls rapidly at the rate of 1,000,000 to 2,000,000 in twenty-four hours. The rate of hemolysis may be extreme in the severe acute crisis, the hemoglobin falling to as low as 4 gm. and the erythrocytes to less than 1,500,000 cells per cubic millimeter in thirty-six hours or less.

Symptoms of hemolytic jaundice usually appear within the first three decades of life. Pemberton,[13] in his report on 118 patients treated by splenectomy, mentioned that 21 patients underwent operation in the first decade of life. Thirty-eight patients were between the ages of 20 and 30 years when operation was performed. Sharpe[14] reported a range of ages from 3 to 63 years at the time of diagnosis, in a series of 28 cases. No sex or race predilection has been claimed.

LABORATORY DATA

Whereas, on the basis of clinical data alone, familial hemolytic jaundice might well be confused with other diseases of the liver, gallbladder, or spleen, the diagnosis can hardly be mistaken when a combination of characteristic laboratory data is added. Anemia is the most obvious and usually the first condition to be encountered. It varies from mild anemia in the latent form to marked anemia in cases of severe crisis.

Blood smears demonstrate small, thick red blood cells or spherocytes which vary in number from case to case and from time to time in the same case. Although variable in degree, reticulocytosis is present constantly. McLaughlin and associates[7] reported from 5 to 30 per cent reticulocytes instead of the normal 0.1 to 0.5 per cent. The increased fragility of the erythrocytes to hypotonic saline solution is a characteristic finding but not necessarily a constant one. Dawson[6] stated that of his 40 patients, 5 did not have increased fragility of red blood cells. Thompson[15] reported the case of a youth who had all the symptoms of familial hemolytic icterus except increased fragility. This patient was completely relieved of all symptoms by splenectomy.

Hyperplasia of the bone marrow, reflected by the increased number of reticulocytes, is confirmed by sternal aspiration which usually shows erythrocytic hyperplasia, chiefly of the normocytic type. The results of the indirect van den Bergh test are positive. Urobilin is present in the urine. When the common bile duct is concurrently obstructed bile is present in the urine and the stool is clay colored.

DIAGNOSIS

The diagnosis of acute familial hemolytic jaundice has, in the past, frequently been confused with diseases of the biliary system,[16] and treatment has been directed toward the gallbladder and the common duct. In Pemberton's[13] series, 23 of 118 patients had had operations on the biliary tract performed elsewhere without knowledge of the presence of the primary disease.

COMPLICATIONS

The most commonly encountered complication of familial hemolytic jaundice is pigmentary cholelithiasis. Sharpe[14] reported that 63 per cent of 14 patients had stones in the gallbladder, while about 69 per cent, or 81 of 118 patients, in Pemberton's series had conclusive evidence of disease of the gallbladder with or without stones. A simple, yet acceptable explanation is that the persistently elevated level of bilirubin in the blood and the resultant increased excretory function of the liver and gallbladder result in an abnormal concentration of pigment in the bile. Precipitation of these pigmentary constituents[17,18] may occur in the gallbladder or even as bile thrombi in the liver. These bile thrombi[19] may later break off and serve as nuclei for stones in the gallbladder or bile passages. In some instances precipitation of soft pigment sludge or formation of stones in, or migration of stones into, the common duct results in its obstruction. The resultant obstructive jaundice, superimposed on a pre-existing hemolytic jaundice presents a complicated clinical picture and a diagnostic problem.

Among other complications encountered are marked acute exacerbations of latent hemolytic disease seen during pregnancy and the rare but troublesome chronic ulcers[20] affecting the lower parts of the legs and the feet.

TREATMENT

Thirteen years before Minkowsky published his description of hemolytic jaundice Spencer Wells[21] performed the first recorded splenectomy on a jaundiced patient who had a large spleen, without fully understanding the condition with which he was dealing. Forty years later Dawson[6] examined the woman and found that she had enjoyed per-

fect health since splenectomy was performed. Examination showed that the erythrocytes had retained their characteristic fragility.

For many years, splenectomy has been accepted as the treatment of choice for permanent clinical relief of symptoms due to hemolytic jaundice.[6, 13, 22, 23] Reasons for the beneficial results which have been obtained have remained obscure. They are, however, of academic interest only in light of the fact that after splenectomy, the accelerated destruction of erythrocytes slows down to normal and as a consequence jaundice eventually vanishes. The number of red blood cells and reticulocytes assumes normal values. The patient regains strength and acquires vitality and a sense of well-being not previously enjoyed.

If the disease is ascertained to be of the latent type, and normal existence of the patient is not disturbed splenectomy may not be necessary. On the other hand patients having the chronic form with acute exacerbations of varying degrees of severity should, in the opinion of most authorities, be advised to undergo splenectomy, since only by this treatment can fatality be avoided in a certain percentage of cases.

In cases of acute hemolytic crisis, when the condition of the patient is grave, splenectomy is likewise advisable. Doan, Curtis, and Wiseman[11] in 1935 reported good results from emergency splenectomies performed when the red blood cells numbered 1,000,000 or less.

Transfusion of blood during acute hemolytic crisis[6, 10, 24] is debatable. It is generally agreed today that even with very carefully cross-matched blood, transfusions may result in a severe reaction with increased hemolysis and further drop in the number of red cells. Blood may be given safely as soon as the splenic pedicle has been ligated. McLaughlin and his co-workers[7] offered the opinion that transfusions of blood are infrequently needed because of the autotransfusion that results from splenic manipulation at the time of operation.

As previously stated, the typical symptom complex of familial hemolytic jaundice easily may be misdiagnosed as biliary disease. The high incidence of gallstones associated with this disease, on a mathematical basis alone, would lead one to expect rather frequent simultaneous attacks of biliary colic and hemolytic crisis. This possibility has been mentioned frequently by writers on the subject, but few[22, 23, 25] have cited cases where the conditions occurred simultaneously.

Although the literature was not exhausted in preparation of this paper, reference was not found to any case similar to the one which we encountered at the Mayo Clinic; that is, a case in which the patient had a familial hemolytic jaundice in crisis, complicated by cholelithiasis, acute purulent cholecystitis with gangrene and perforation, and was treated successfully by combined splenectomy and cholecystectomy.

REPORT OF CASE

A married, white woman, 27 years of age, was hospitalized in an emergency on July 21, 1948. Her chief complaints were pain and jaundice.

Inquiry concerning her illness revealed that in 1940 she had experienced sudden onset of painless jaundice, anorexia, and vomiting. She did not take her temperature but believed that it was elevated. She recalled that her father and brother were ill with identical complaints at the same time. Her stools were normal in color, but she did not note the color of her urine. The attack gradually subsided over a period of three weeks. Six years later she had an identical attack, with the exception that this time she did not have fever and she noted that her urine was a darker color than normal.

During the course of her second pregnancy which terminated with normal spontaneous delivery on May 10, 1948, it was noted that she was anemic. Liver extract was administered parenterally; iron tablets and liver and iron capsules also were administered.

One month prior to admission to the hospital, the patient was awakened by severe pain in the epigastrium. The pain which was so intense that several hypodermic injections of morphine sulfate were required for relief, migrated posteriorly and settled beneath the right scapula. She had chills and vomited once. Jaundice again appeared. The urine became darker but the stools remained of normal color.

Her condition again improved gradually, with abatement of jaundice and subsidence of pain until she experienced a sudden recurrence of steady, intense subscapular pain one week before she came to the clinic. This time the pain persisted, and repeated doses of narcotics were required. The icterus was intensified, the urine became darker once more, and the stools again were normal in color.

The patient's history was noncontributory except for an intolerance of many years standing for fried foods, gravies, pork, cabbage, and onions.

The patient was obese, moderately jaundiced, and appeared acutely ill. Blood pressure was 100 systolic and 50 diastolic expressed in millimeters of mercury. The pulse rate was 110 beats per minute and the temperature was 101° F. Complete physical examination disclosed icterus of the skin, grade 2, icterus of the sclerae, grade 2, and pallor, grade 3 (all grading on a basis of 1 to 4, grade 1 being the least degree and grade 4 the greatest). A harsh systolic murmur was heard over the entire precordium. The spleen was palpable a distance of 5 or 6 fingerbreadths below the left costal margin. On palpation there was tenderness, grade 2 in the right upper quadrant.

Initial laboratory data are given in Table 1. Blood smears were reported as typical of congenital hemolytic jaundice. A sternal puncture yielded a few units of marrow, mostly small. Microscopically, there was evidence that regeneration of both myelocytes and erythrocytes was active. Results of the Kline test were

TABLE 1
FAMILIAL HEMOLYTIC JAUNDICE: LABORATORY DATA

	ADMISSION	THIRD HOSPITAL DAY	TWENTY-FIRST DAY AFTER OPERATION	SIX MONTHS AFTER OPERATION
Hemoglobin, gm.	4.8		9.1	13.9
Erythrocytes, millions per cu. mm.	1.78		3.10	4.84
Leukocytes, per cu. mm.	14,000		9,900	11,300
Differential count				
Lymphocytes	20			
Monocytes	5			
Neutrophils	75			
Filamented	59			
Nonfilamented	16			
Reticulocyte count	9.8%		0.2%	0.4%
Red blood cell, fragility	0.50-0.36%			0.50-0.28%
Sedimentation rate (Westergren), mm. per hr.	164			
Serum bilirubin, mg. per cent				
Direct	2.69	14.1	0.6	0
Indirect	1.40	5.0	0.5	0.8
Bromsulfalein liver function test, per cent dye retention			0	0
Blood smear	Typical of familial hemolytic jaundice			General microcytosis. Some spherocytosis

negative. The blood was of group A with a positive Rh factor. A voided specimen of urine showed albuminuria, grade 3, and 1 red blood cell, and 5 pus cells per field. There was no bile in the urine. A roentgenogram of the thorax revealed rather marked generalized enlargement of the heart.

The patient's temperature continued to spike upward and reached a peak of 104.2° F. early in the morning of the third hospital day. At this time she was experiencing severe pain in the right subscapular region and a marked increase in jaundice was evident. The serum bilirubin rose to 14.1 mg. per 100 cc. direct and 5.0 mg. per 100 cc. indirect (Table 1).

With these developments it was felt that the possibility of obstruction of the common duct by stone should be added to the earlier diagnosis of congenital hemolytic jaundice in crisis, complicated by cholelithiasis. It was decided that splenectomy should be performed and that the gallbladder and the common duct should be explored at the same time, if possible.

ACCORDINGLY, on July 23, 1948, after premedication consisting of 72 mg. of menadione bisulfite, administered intravenously, 1½ gr. (0.1 gm.) of pentobarbital sodium given orally, 1/6 gr. (0.01 gm.) of morphine sulfate and 1/150 gr. (0.00043 gm.) of atropine sulfate given intramuscularly, the patient was anesthetized with nitrous oxide, oxygen, and ether.

An exploratory laparotomy was performed, through a primary left Kocher incision. The spleen was found to be greatly enlarged, being approximately 10 by 5 by 5 inches in size. It was mobilized with moderate difficulty because of the patient's obesity. Splenectomy was performed, and the splenic artery was ligated with double chromic catgut No. 0 and with a stick tie of silk in addition. The splenic bed was peritonized, and the border of the pancreas was explored for accessory spleens but none were found. The uterus and left ovary were normal in size. A thin-walled cyst, about 2 inches (5 cm.) in diameter was located on the right ovary.

The region surrounding the gallbladder was explored and an indurated mass completely covered by adherent omentum was revealed. While this region was being palpated, some of the omentum was separated, and pus and gallstones were encountered. Exposure was then improved by extending the initial incision 3 inches (about 8 cm.) along the right costal margin. The omentum separated from the gallbladder easily, and 2 perforations were disclosed in the fundus of the gallbladder.

The gallbladder was then evacuated and subsequently freed from its bed in a retrograde fashion, by means of blunt dissection. Because the gastrohepatic ligament could not be demonstrated satisfactorily the gallbladder was divided at its ampulla after double ligation with chromic catgut. A small rent in the duodenum, which occurred while separating the

duodenum from the ampulla of the gallbladder to which it had adhered, was repaired with interrupted silk sutures. The gallbladder bed was peritonized. Five gm. of sulfathiazole powder was left in the right upper quadrant of the abdomen and 3 Penrose drains from this area were brought out through a stab wound in the midline. The abdomen was closed in layers.

The pathologist's report was splenomegaly, grade 4, on the basis of 1 to 4, with hemosiderosis. They found microscopic observations to be consistent with those of congenital hemolytic icterus.

A transfusion of 1,000 cc. of blood was started as soon as the splenic vessels were ligated, and an additional transfusion of 500 cc. was given as a supportive measure immediately after operation. Antibiotics in the form of penicillin, 160,000 units a day for a total of 1,280,000 units, and streptomycin, 1.6 gm. each day for a total of 12.8 gm. were given intramuscularly through the eighth postoperative day.

Postoperatively the temperature rose gradually and reached a peak of 104° F. on the fourth day; it then fell rapidly until the seventh day when it was a normal level.

There was considerable drainage from the stab wound, bile-stained at first, but after the first week, serous in character. All drains were removed on the thirteenth postoperative day and the patient was dismissed from the hospital on the fourteenth postoperative day.

Laboratory examinations were made on August 11, 1948, the twenty-first postoperative day (Table 1).

At the time of dismissal from the clinic on the twenty-third postoperative day, the patient felt strong and energetic and had no distress. Both the urine and the stools were normal in color.

Six months later, on February 15, 1949, the patient returned for a postoperative check-up. She had been feeling well and had gained weight. She had had no abdominal pain or discomfort in the interim and had been able to eat any food she desired. She had not experienced any chills, fever, or jaundice. Stools and urine had been normal at all times. The abdominal incision was well healed and strong.

Laboratory examinations again were made at the time of the return visit (Table 1). A blood smear showed the evidence of generalized microcytosis with some spherocytosis; erythrocytes measured by means of the erythrocytometer averaged 6.6 microns. Howell-Jolly bodies were present. The direct reading for serum bilirubin was negative; the indirect reading was 0.8 mg. per 100 cc. The bromsulfalein test for liver function showed 0 per cent dye retention after one hour. Increase in fragility of erythrocytes again was observed; hemolysis began in 0.50 per cent and was completed in 0.28 per cent saline solution.

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GENERAL PRACTICE *Review*

To provide the general practitioner, as well as the specialist with concise, readily available information on the latest, proved methods of treatment of conditions commonly encountered in an average practice, POSTGRADUATE MEDICINE *offers this department to its readers. It is not, of course, intended to present these discussions as the only acceptable therapeutic procedures to be used, but rather to offer simple regimens and recommendations based on the extensive experience of the physicians who prepared these summaries.*

Migraine Headache: What Can Be Done About It?

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MIGRAINE headache results from widening and stretching of the arteries of the head, chiefly those on the outside of the skull. Many people can momentarily help their headache by pressing at the site of the pain. Cold applications make the vessels narrower and may help reduce the pain. Any of the arteries of the head can be affected and produce headaches, but those in the region of the temples are most commonly involved.

Because these headaches may vary from a dull one-sided ache to a pounding, prostrating pain involving the entire head, several means to relieve them can be used, depending on the severity. A mild or moderate headache can be relieved by two aspirins and a cup of strong coffee, or one or another of the common headache remedies. Narcotics other than codeine should be avoided because of the danger of addiction in this recurrent disorder.

When the pain is severe, and the headache cannot be relieved by agents which reduce pain, a drug which narrows the painfully stretched arteries is used. This drug—ergotamine tartrate (Gynergen) in doses of 0.25 to 0.5 mg. given as soon as possible after onset of pain—will usually cause the headache to diminish or disappear within an hour after injection. Unfortunately it cannot be used too frequently without harm but can be given once or twice a week to healthy patients. Dihydroergotamine (DHE 45) is somewhat less predictable in doses of 1 to 2 mg. It may produce less nausea and vomiting. Pregnancy, vascular disease, liver disease, and hypertension are contraindications to use of the ergot derivatives.

It is important for the patient to lie down as soon as possible after the beginning of a headache, preferably in a darkened, quiet room. An ice cap to the painful part of the head and a heating pad or hot water bottle to the abdomen or feet are helpful. Sodium amytal, 0.2 gm., helps reduce nausea and promotes needed rest.

WHY DO MIGRAINE HEADACHES OCCUR?

What causes these head arteries to stretch so painfully? The answer is—meeting life with attitudes and points of view that produce worry, fear, tension, resentment, rage, and exhaustion. These are building-blocks of the common sick headache.

The patient with migraine is not necessarily "neurotic" in the usual sense; he may, in fact, have made a very successful adjustment in life. However, these people have certain personality characteristics in common. They have the defects of their qualities.

They are tense, driving people who are rigid, ambitious, and perfectionistic. They have found that doing "more than and better than" their fellows brings considerable satisfaction but at great cost in energy. They are considered conscientious and hard working, and because of this, more and more responsibility comes their way. Because they delegate responsibility poorly, they become resentful because they cannot keep up with the load which the world and they themselves impose. As a result, tension, fatigue, and exhaustion mount, and in this setting headache makes its appearance.

HOW CAN HEADACHES BE CURED?

Nothing out of a bottle will offer more than passing relief. "Cure" must be brought about by the patient himself, guided by his doctor. Cure of headache really means prevention of headache. In order to accomplish this, habits of living and habits of thinking must be changed.

Reduce the day's work—One of the most important changes is to cut down on the number of things ordinarily done in a day. The patient must learn to be less fussy about minor details.

Everyone has days when he feels tired and irritable and finds his work harder to do. This is true of women during their menstrual periods. Attempting to drive one's self to do as much on "tired" days as on "energetic, vigorous" days results in much fatigue and tension and very often produces headaches. He must recognize this and deliberately plan to do less on such days. A person should not feel frustrated because less has been accomplished, because lost time will be made up on better days.

As a person grows older he becomes more easily tired. One should not expect to do as much at the age of 50 as at 20.

Time should be allowed for enough rest. At least eight hours of sleep every night is essential. A half hour's rest after lunch provides a useful break in the day's activities. A good way to learn to rest and relax is to take a warm bath after returning from work, or whenever tired and tense. Fill the tub with warm water and put a support under the head. Lie in the tub and relax completely for half an hour.

Some exercise should be included in the daily program. Many city dwellers find an evening walk relaxing and enjoyable. Any other kind of moderate exercise which is fun is of immense help in reducing tension. Fun and a pleasant sense of "tiredness"—not exhaustion—are the goals.

Avoid needless and useless worry—Worry seldom really solves anything and only makes one tired and tense. A person who has headaches must not allow himself this needless waste of energy. Put the cat or the mother-in-law out. Lying in bed worrying whether or not to do so will not help.

Change some of your standards—The most important thing about curing headache is to change ideals and standards to fit in better with everyday experiences. Each person has to do this for himself. If standards are needlessly high, they must be changed. If rigid ideals are not adding to enjoyment and satisfaction in life, it would be foolish not to change them. It is important to change attitudes about one's own high standards and their lack in others so that resentment, anxiety, disappointment, and fatigue do not develop. It is essential to get satisfaction out of what one can have and can do.

Develop tolerance—Particularly important is *fuller tolerance* of other people. Husbands, wives, and friends may not fit the ideal, but they all have good qualities nonetheless. Try to understand other people better, learn to see their point of view and to like them for their good qualities. Tolerance about one's job is essential. It is important to be less concerned with insignificant details and to be generous in opinions about fellow-workers. Resentment should not follow if others are not as efficient and industrious as they might be.

The patient must learn to let the virtue of doing things well bring satisfaction instead of anxiety and disappointment. He has to be more tolerant about the home. A dirty dish in the sink or a little dust on the table should not be upsetting. Instead, appreciate the good things of home and enjoy its comforts Happiness must not be destroyed because every little thing is not just so.

These changes in habits of living and thinking will bring a feeling of relief and a new enjoyment of life. The machinery of the body, relieved of tension, runs more smoothly and efficiently, and illness is avoided.

Every headache must be made to pay its way in terms of increased understanding. The important thing is to make the headache into a *useful lesson*. Each headache means only one thing—failure to profit by what he has learned about his way of meeting life's situations. Each time the patient fails, it is up to him to figure out why. An honest, understanding effort to improve ways of thinking, feeling, and doing will certainly be rewarded by more satisfaction in living and by longer and longer periods of freedom from headache.

Modern Principles of Hand Surgery

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Proper initial treatment of a hand injury pays heavy dividends in ultimate function. A careful sensory and motor examination prior to operation will determine accurately the extent of surgical repair needed. If flexor tendons or nerves are involved special surgical technics are necessary for successful results; if these are not immediately available, simple debridement and suture of the wound is in order, leaving anastomoses for a secondary procedure under optimal conditions.

Certain operating room technics should rightfully be incorporated in office or clinic practice. A sturdy armboard supported by a leg and the routine use of a tourniquet make minor surgical procedures much easier and more accurate. Nerve block at the wrist permits painless irrigation and debridement of hand wounds and is particularly valuable in the treatment of minor localized infections which should not be injected directly, and which otherwise would require general anesthesia and hospitalization.

Even small losses of skin should be treated initially by covering with free split-thickness grafts, thus hastening the healing and minimizing the danger of contractures. The use of a graft will not necessitate hospitalization unless the wound itself warrants it and will usually reduce the expense of convalescence materially.

The operative dressing is important in shortening convalescence. Most wounds deserve a pressure dressing to the affected part, combined with immobilization by plaster of the adjacent joints. The hand should be maintained in the position of function, with the wrist dorsiflexed, the fingers semi-flexed, and the thumb abducted and in opposition to the finger tips (Figure 1). Burned hands should be similarly positioned after decorticating vesicles and supported by a bulky dressing of fluffed gauze or surgical waste.

POSTOPERATIVE CARE

Rehabilitation should be guided by three cardinal objectives: (1) flexion which will give forceful grip and permit the retrieving of small objects; (2) sufficient extension of the digits to permit encompassing of large objects such as a drinking glass; and (3) tactile sensation on the volar aspect of the thumb and at least two adjacent fingers.

Any hand injury, even a minor one, results in the accumulation of inflammatory products which tend to stiffen the digits, particularly when immobilization is prolonged. To minimize inflammatory edema and stiffness all granulating wounds should be resurfaced by free grafts as soon as pathogenic contaminants can be eliminated.

Active motion should be started usually within five to seven days after injury; however, in the case of tendon repair the consensus is that from two and one-half to three weeks of immobilization will produce less peritendinous fibrosis and more rapid return of activity than shorter periods of rest. The importance of differentiated exercise of the digits should be stressed and the patient taught how to exercise each phalanx individually, immobilizing the more proximal part of the digit with the opposite hand.

Physiotherapy in the form of hydrotherapy is a most useful adjunct, either in the office or as a simple soak at home, but the patient must be made to realize that this is only supplemental to his most significant therapy, namely, an hourly program of active exercise carried out methodically and intelligently in graduated manner.

Corrective splints should be introduced immediately if any tendency toward contracture is noted. The most common contractures consist of limitation of extension of the fingers, limitation of abduction of the thumb, and incomplete opposition. Light metal splints providing corrective elastic traction are available commercially or can be fashioned quite easily in the office[1] (Figure 2).

Any splint should be designed so that maximal activity of the hand can be maintained while constant elastic traction is being brought to bear upon the contracted part. If contractures are treated promptly, a night splint is often sufficient to maintain proper range of motion, permitting free use of the hand during waking hours. The night splint is particularly effective in preventing the contraction of grafts during the vulnerable first three months after operation.

Finally it should be emphasized that newer concepts of treatment have significantly increased the percentage of damaged hands that can be restored to useful function. Amputation as a primary operative procedure should be performed only in the most obvious cases of irreparable injury.

The great majority of severe injuries should be treated by complete debridement, primary grafting of denuded surfaces, and immobilization in a functional position. Having preserved and healed the basic structures one may then proceed with reconstructive measures.

FIGURE 1. Position of function in which the hand should usually be immobilized postoperatively.

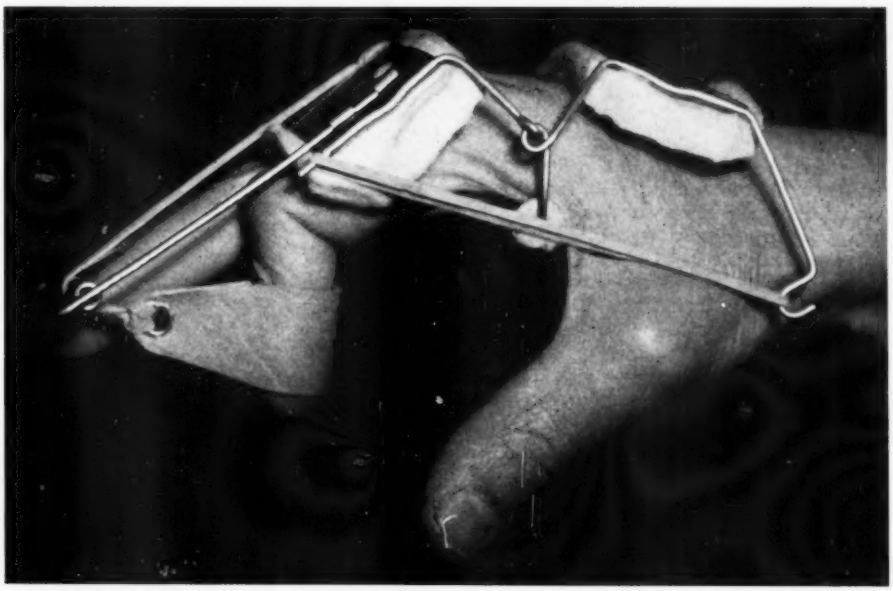

FIGURE 2. Example of dynamic splinting which produces through elastic traction flexion of metacarpophalangeal joints and extension of index finger while permitting free active motion of the hand in general.

Temporary free grafts or scars may be replaced by abdominal pedicle flaps to afford a soft, fatty bed for mobilization of tendons or for repair of nerves. Bone grafts may be used to stabilize or reconstitute metacarpal bones. Extensor tendons of the toes or forearm tendons may be utilized to restore essential digital motion and less important tendon-muscle units may be transferred to perform other functions in cases where irreparable nerve damage has taken place.

Dorsal sympathetic block anesthesia or sympathectomy has produced dramatic changes in causalgic states and has restored to usefulness many hands which otherwise would have remained permanently crippled because of impaired vascularity.

This brief recital should offer sufficient justification for the modern thesis that primary surgery in hand injuries should carry as its keystone the maximum preservation of tissue.

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Treatment of Endocarditis

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There are three types of endocarditis: nonbacterial, acute bacterial, and subacute bacterial. A few general characteristics of each of these types should be known before one enters into the specific measures of treatment.

There are several types of nonbacterial endocarditis but the majority are caused by rheumatic fever. This process often leads to valvular deformities and eventually to what is called chronic rheumatic valvular disease of the heart. The majority of patients recover from this nonbacterial rheumatic endocarditis. The valvular deformities, however, may predispose to still another form of endocarditis in later life, the subacute bacterial form.

Acute bacterial endocarditis, on the other hand, is part of an overwhelming generalized infection with involvement of the blood stream. It is nearly always secondary to some other primary disease process, and in this manner almost any infectious process may at times become the primary cause of the endocarditis. There are certain organisms, however, which are responsible for most cases of acute endocarditis: the hemolytic streptococcus, pneumococcus, staphylococcus, gonococcus, influenza bacillus, meningococcus, and occasionally Brucella and E. coli or other organisms.

It may often be very difficult or even impossible to diagnose this condition in an acutely ill patient. Unlike subacute bacterial endocarditis this disease may affect heart valves which were previously normal.[1] Before the advent of the antibiotics this disease was almost always fatal.

Subacute bacterial endocarditis as a rule presumes prior valvular damage, the majority of which have been caused by a previous nonbacterial rheumatic endocarditis. Congenital heart disease is the basis upon which subacute bacterial endocarditis is superimposed in a smaller percentage of cases. Nonhemolytic streptococcus is responsible for from 90 to 95 per cent of the cases of subacute bacterial endocarditis. The gonococcus, unidentified gram-negative bacillus, and diplococcus as well as the staphylococcus albus and the enterococcus are occasionally the causative organisms.[2] The onset is insidious and the course is prolonged.

Every patient with valvular heart disease should receive prophylactic penicillin before and after any dental as well as other surgical procedures. The usual method for carrying out this protection is by giving at least 300,000 units of one of the slower acting forms of penicillin the day before, the day of, and the day following these procedures.

SPECIFIC MEASURES

Nonbacterial endocarditis—Suffice it to say that for the present there is no specific form of treatment for this condition. Since the majority of these cases fall into the rheumatic fever group they will be treated by the usual accepted methods employed for this disease.

Acute bacterial endocarditis—The treatment of this form of endocarditis will of course depend upon the organism causing the generalized infection. The majority of the frequently offending organisms—hemolytic streptococcus, pneumococcus, staphylococcus, gonococcus, and to some extent the meningococcus—fortunately are sensitive

in varying degrees to penicillin. Sulfadiazine, however, is still the drug of choice in meningococcic infections. The colon bacillus usually responds to the sulfonamides and streptomycin. The brucella organism gives promise of yielding to aureomycin or to aureomycin plus streptomycin.

Only by keeping the condition in mind and obtaining early blood cultures will the true status be proved early enough to plan therapy. If time permits, in vitro sensitivity tests should be done. Usually one must depend upon clinical judgment to determine the dosage but it should be high at the onset and adjusted later depending upon the clinical response and results of in vitro sensitivity tests.

Subacute bacterial endocarditis—Reports on the treatment of this condition by many varied and nonspecific measures prior to the advent and availability of penicillin in 1944 were very pessimistic. Paullin and McLoughlin in reviewing such reports in 1945 stated that in a collection of 2,600 cases there were only 25 spontaneous recoveries.[3] Kelson and White reported on 250 cases in 1945 in which all but one succumbed to this dreaded disease.[4] Favour, Janeway, Gibson, and Levine in 1946 reported 237 cases treated between 1913 and 1937 in which no recoveries were known.[5]

Fortunately, most strains of the streptococcus viridans are sensitive to penicillin. This antibiotic has had a profound effect upon the outcome of this disease, reducing the mortality rate from well over 90 per cent to approximately 30 per cent. Not enough experience has accumulated concerning the effects of streptomycin, aureomycin, and chloromycetin to predict how much lower this mortality rate will fall with their addition; however, it is believed they will certainly render valuable aid.

The first step in planning a course of therapy after the organism and strain have been identified is to determine the in vitro sensitivity of that particular organism. Accumulated experience indicates that the best policy is to establish a blood concentration of penicillin which is 3 to 5 times the in vitro susceptibility of the organism. Some authorities state that no patient regardless of the in vitro sensitivity should receive less than 500,000 units per day for at least four weeks.

The resistant streptococcus faecalis (enterococcus) may resist dosages of several million units per day. Dosages of as high as 75,000,000 units per day have been employed in treating this strain. A small number of cases of this condition have responded to the combined use of large dosages of penicillin plus streptomycin up to 4 gm. per day. When high dosages of penicillin are desirable, caronamide may be a valuable adjunct since it slows the rate of excretion of penicillin by the kidneys.

The modes of administration have been the intermittent intramuscular, intermittent intravenous, continuous intramuscular, and continuous intravenous methods. In order to administer very large dosages, all of these methods may be necessary. The choice will depend somewhat upon the form of penicillin used and the desired blood concentration. When the aqueous solution is employed and very high concentrations are desired the continuous intramuscular route is probably the choice procedure and next, frequent intermittent intramuscular injections. When the slower-acting forms of penicillin are employed and the dosage is not to be extremely high, the intermittent intramuscular method would probably be the method of choice.

The intravenous methods have a disadvantage in that chemical thrombophlebitis occurs at or near the site of injection in a high percentage of those treated in this manner. The time interval will also depend upon the above factors.

Relapses may occur especially if the therapy is discontinued before four to six weeks of continuous treatment have been completed. The majority of these occur within the first four weeks after cessation of treatment. No patient should be considered cured until he has been free of the signs and symptoms of the disease and his blood cultures have been negative for at least six weeks after the conclusion of therapy. If a relapse occurs, the in vitro sensitivity should again be determined before treatment is resumed, as the resistance of the organism may change after the previous therapy.

Emboli are a constant threat at any time during the activity of this disease. They may occur after the blood stream has been rendered sterile and do not necessarily constitute a relapse of the disease.

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Scientific Exhibit

Antihistamine Therapy in Dermatology

GEORGE M. LEWIS, M.D., JEFF DAVIS, M.D., AND
GEORGE A. WALDRIFF, M.D.

CORNELL UNIVERSITY MEDICAL COLLEGE, NEW YORK CITY

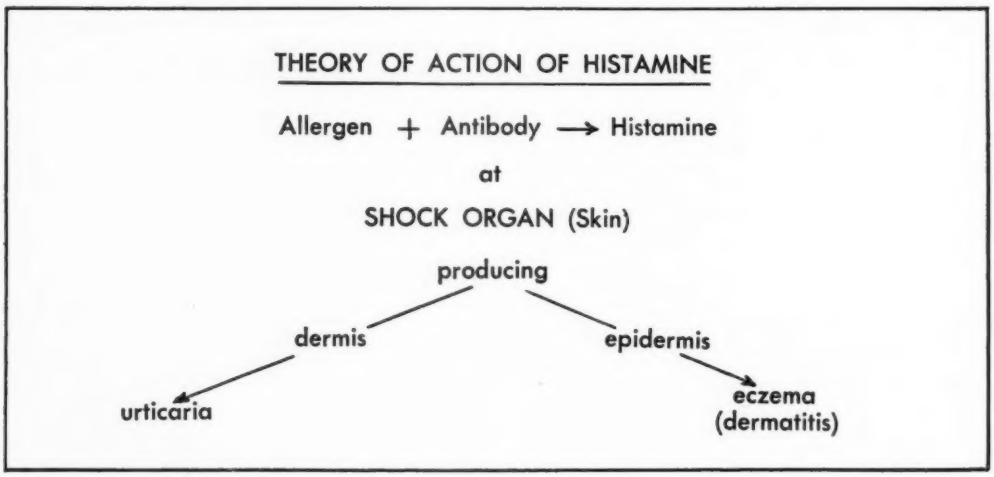
THE antihistaminic drugs are useful adjuncts to the therapy of many skin diseases. Subjectively, there is often relief from pruritus. Objectively, the eruption may be favorably influenced.

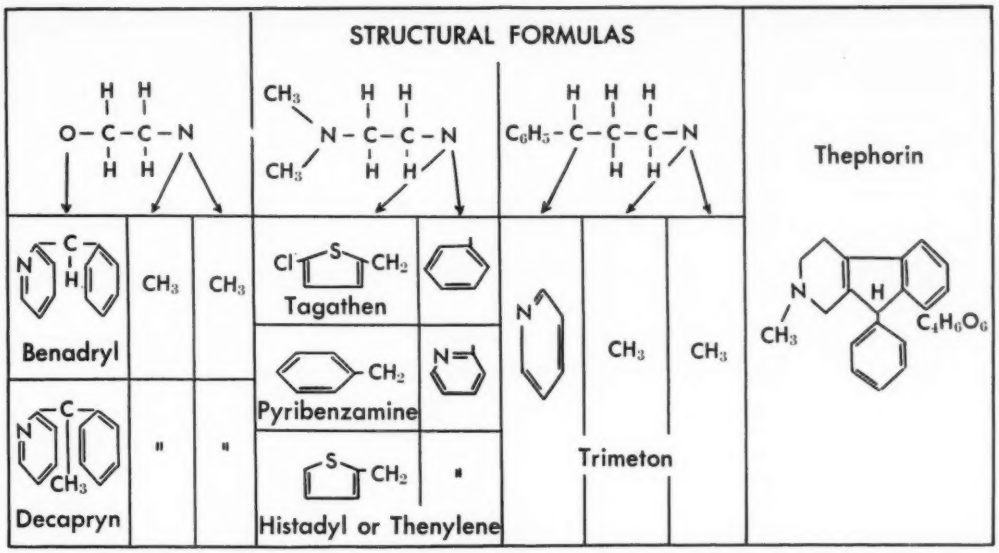
Since 1910 when Dale and Laidlaw first advanced the histamine hypothesis as a factor in allergic disorders, many workers have contributed to the development of our present knowledge. The first antihistaminic drugs were developed in 1937 in France by Bovet and Staub. Since then, many new compounds have been synthesized and a comparative study of the clinical effectiveness of eight of these chemicals is herewith reported.

Our project included the administration of the drugs by the oral route and by application topically. The medications were given alone or in combination with standard and accepted methods.

A wide range of pruritic dermatoses was treated experimentally. When used internally, the drugs were usually prescribed in a dosage of 25 or 50 mg. three or four times daily. Occasionally, an increase in the dose was required before a therapeutic effect was obtained. Combinations of two or more antihistaminic drugs were given to some patients. Reactions were observed in 24 per cent of our patients but were not serious. Because of the wide selection, it was always possible to complete therapy using a product which did not cause reaction.

The drugs are often effective when applied directly to pruritic skin lesions, exerting a procaine-like effect. However, antihistamine drugs should not be applied to exudative or abraided surfaces. In such patients, the evidence of local reaction was 22 per cent. The average incidence, in all patients for irritation or sensitization dermatitis, was found to be 5 per cent.





THEORIES OF ACTION OF ANTIHISTAMINES

1. Prevents release of histamine
2. Neutralizes histamine after it is formed
3. Competes with histamine for chemical combinations with the cell
4. Stimulates sympathetic system (adrenergic) and thus opposes the pharmacologic action of histamine

ORAL THERAPY

COMPARATIVE DATA ON ORAL ANTIHISTAMINES—389 CASES

Drug	*No. of Drug Trials*	*Number Benefited*	*Per Cent Benefited*	*Number of Side Reactions*	*Per Cent of Side Reactions*
Thephorin	258	151	59	64	(25) 18*
Neo-Antergan	96	62	64	19	20
Tagathen	64	43	67	17	(22) 18*
Trimeton	144	100	70	34	(24) 19*
Benadryl	46	17	37	20	43
Pyribenzamine	105	40	38	20	19
Decapryn	51	28	55	9	18
Histadyl	30	17	56	7	23
	794 *total*	458 *total*	58 *average*	190 *total*	24 *average*

**Result when dose was reduced from 50 to 25 mg. three times a day.*

ORAL THERAPY

SIDE REACTIONS

Of 345 patients who received an antihistamine drug by the oral route, 147, or 20 per cent, had one or more side reactions as listed below. There was an average of two complaints for each reactor.

Physical and laboratory examinations failed to reveal any pertinent findings and on withdrawing the drug the symptoms disappeared promptly.

The ten most commonly observed reactions are numbered in order of their frequency.

Central Nervous

5 Headache
1 Drowsiness
3 Restlessness
Depression
2 Dizziness
Excitement
Diplopia
Sweating
Irritability
6 Insomnia
Nervousness
Paresthesia
Increase in itching
Blurred vision
Dilated pupils
Euphoria
Weakness

Respiratory

Respiratory distress

Gastrointestinal

4 Nausea
Vomiting
7 Anorexia
Indigestion
Abdominal cramps
9 Constipation
Diarrhea
Metallic taste
10 Dryness of mouth

Genitourinary

Frequency
Impotency

Cardiovascular

8 Palpitation
Precordial pain
Tachycardia

Cutaneous

Erythema, multiforme-like

EFFECTIVENESS BY DISEASE GROUPS

Disease	*Number Drug Trials of Antihistamines Alone*	*Per Cent Obtaining Relief*	*No. Drug Trials Combined Method*	*Per Cent Obtaining Relief*	*Average Per Cent Both Groups*
Urticaria	180	80	0	0	80
Neurodermatitis circumscripta	47	64	50	66	65
Disseminated neurodermatitis	142	58	39	62	58
Contact dermatitis	38	47	47	38	42
Pruritus ani, vulvae or scrota	20	25	15	67	43
Miscellaneous	139	47	78	37	43
	566 *total*	61 *average*	229 *total*	50 *average*	58 *average*

EFFECTIVENESS BY DISEASE GROUPS		
Disease	*No. of Cases*	*Per Cent Effective*
Neurodermatitis circumscripta	76	82
Pruritus ani, vulvae, scrota	74	79
Neurodermatitis disseminata	37	74
Contact dermatitis	29	64
Miscellaneous pruritides	61	38
	277 *total*	72 *average*

REACTIONS TO TOPICAL TREATMENT

An irritating or sensitizing effect may be expected in 4.5 per cent of patients when an antihistamine drug is applied directly to a skin disease. The reaction is not usually severe if the incompatibility of drug for skin is quickly recognized. A tingling or burning sensation is frequently experienced for several minutes following the application of the drug. The antihistamine drugs should not be applied directly to exudative dermatoses.

PATCH TESTS

In order to ascertain the vulnerability of the general population to irritation and sensitization from local contact to these drugs, a series of patch tests was performed on the normal skin of 100 subjects using 7 different antihistamine compounds each in the concentrations commonly advised for therapy. The patients were retested in from ten to fourteen days with the same preparations. Control applications included the ointment bases supplied by the manufacturers. There were 13 moderate irritation reactions among 8 patients, an incidence of less than 1 per cent. One patient developed an irritative dermatitis from an ointment base. On retesting, 3 patients revealed sensitization to a total of 5 products.

COMPARATIVE DATA				
Drug	*No. of Drug Trials*	*Per Cent Effective*	*No. of Reactions*	*Per Cent Reactions*
Thephorin	194	83	8	4
Histadyl	66	79	2	3
Decapryn	38	45	1	2
Benadryl	46	65	2	4
Pyribenzamine	143	66	9	6
Trimeton	94	78	2	2
Thenylene	63	73	5	8
	644 *total*	72 *average*	29 *total*	5 *average*

EDITORIALS

Postgraduate Medicine OFFICIAL JOURNAL OF THE INTERSTATE POSTGRADUATE MEDICAL ASSOCIATION OF NORTH AMERICA — ORGANIZED IN 1915

Editor-in-chief: CHARLES W. MAYO, M.D.
Contributing Editor and Editorial Writer: MORRIS FISHBEIN, M.D.
Assistant Editor: SYLVIA COVET

MAY 1950

STREPTOMYCIN IN TREATMENT OF TUBERCULOSIS

SINCE 1946, representatives of the Army, Navy and Veterans Administration have been studying the use of streptomycin in treating various forms of tuberculosis. In November, 1949, these investigators held their Eighth Streptomycin Conference. The results presented included reports from 42 hospitals in which streptomycin was used on some 7,000 patients by 22 different plans of treatment. The conclusions, as expressed in a report to the Council on Pharmacy and Chemistry of the American Medical Association, indicate that "in and of itself, streptomycin can never ('well, hardly ever') be counted on to cure a case of tuberculosis." Relapses occur in about 40 per cent of cases of pulmonary tuberculosis and in from 5 to 10 per cent of cases of tuberculosis of the alimentary tract and of the skin, when there are draining sinuses.

The investigators were generally agreed that chemotherapy should be combined with other forms of treatment, including particularly early collapse or excisional surgery when indicated by the patient's condition.

The report summarizes the present status of treatment of tuberculosis with streptomycin by pointing out that the incidence of toxic manifestations can be diminished significantly by reducing dosage. The development of resistance in the bacilli can be only partially avoided by decreasing the duration of treatment to forty-two days but in such practice, with a definite loss of efficiency.

In general, lesions of the mucous membranes of the respiratory and alimentary tracts, draining cutaneous sinuses and peritoneal tuberculosis respond most promptly and uniformly to treatment with streptomycin. A jury of orthopedic surgeons concurred that improvement occurred more rapidly and to larger extent in cases of tuberculosis of bones and joints in patients who received streptomycin than in those who did not receive the drug. Similar usefulness was demonstrated in genitourinary tuberculosis. Streptomycin can save about half of the patients with miliary tuberculosis and about one-fifth of those with tuberculous meningitis. Unfortunately the damage done to tissues of the nervous system is terrific and the residual scarring, destruction of basal ganglia, and hydrocephalus often leave patients who survive in a most unfortunate state.

The investigators took a rather dim view of the value of dihydrostreptomycin. Neomycin is regarded as still too toxic to justify use on human cases of tuberculosis. Paraminosalicylic acid (called PAS) has proved promising when used with streptomycin particularly in cases in which the bacilli have developed resistance to the latter drug.

The continuing reports of the conferences of investigators who are making these studies indicate the great values inherent in such joint and cooperative investigations, especially when properly planned and organized. By this technic the time lost in determining the actual value of new drugs, their potential harmfulness, and the most efficient methods of administration, can be saved or minimized. For this purpose the Committee on Therapeutic Trials was organized. Its inability to be more useful is not due to a weakness of the idea, nor to inadequacy of the Committee but to failure of manufacturers and medical institutions to utilize its services more fully.

MORRIS FISHBEIN

THE LANGUAGE OF PSYCHIATRY

The new terms that come to view first in contributions to the literature of psychiatry are disturbing to physicians in general who try to keep abreast of advancement. In an address at the meeting of the British Psychological Society on "Some Problems of Psychoneurology and Psychological Theories," Robert H. Thouless discussed whether we should use terms current in our speech, such as "intelligence" and "repression," redefining them in the ways necessary, or whether psychologists should construct a purely technical vocabulary, the terms of which they would both create and define.

Examples of purely technical terms are Freud's "cathexis" or from the positive point of view "the general intellectual factor." Early investigators tried to use the terms current in ordinary speech. They did not apparently foresee that the use of such terms in a manner different from that of ordinary speech would be confusing, nor did they see the ultimate possibility that psychologists would have to spend a large part of their time in their meetings in disputes about the meanings of words. Apparently there is no uniform understanding of such terms as "instinct" or "personality." As an example of misunderstanding, Professor Thouless calls attention to such phrases as "intelligence reaches a maximum at 15 years of age" or "the general intellectual factor reaches a maximum at 15 years of age." The man on the street will misunderstand both these phrases. The psychologist is likely to say that he considers the word "intelligence" as meaning something quite different from that supposed by his hearer.

A more interesting example is the statement that "Freud taught that every psychoneurosis has its origin in the repression of a sexual wish." One discovers, however, that Freud did not use the words "repression," "sexual" or "wish" as they are used by people generally. The average person thinks of "repression" as stopping someone from doing something he wants to do. Freud explains it as follows, "The ego drew back, as it were, after the first shock of its conflict with the objectionable impulse; it debarred the impulse from access to consciousness and to direct motor discharge, but at the same time the impulse retained its full charge of energy." The public drew from the expression that repression leads to neurosis the meaning that children become neurotic because their parents do not let them do what they like. Actually Freud meant nothing of the kind.

One gains little by looking in either medical, psychological or ordinary dictionaries to determine the meanings of these terms because those who make the definitions are themselves somewhat at a loss as to the meanings of the words when used out of context. Obviously the leading societies in this field should assemble a general committee to standardize their nomenclature.

MORRIS FISHBEIN

THE ETHICS OF THE PHYSICIAN

Among the most important formulations of the World Medical Association is its International Code of Medical Ethics, adopted at the session held in London, October, 1949. Here in the scope of a few succinct sentences is a practical guide to the medical profession in its relations with the sick and with each other. A series of principles such as this might well be memorized by every senior medical student. With these principles the World Medical Association has incorporated its Declaration of Geneva, a modern oath for the medical profession, which was adopted in Geneva, Switzerland, in 1948.

INTERNATIONAL CODE OF MEDICAL ETHICS

Duties of Doctors in General

A doctor must always maintain the highest standards of professional conduct.

A doctor must not allow himself to be influenced merely by motives of profit.

The following practices are deemed unethical:

(a) Any self-advertisement except such as is expressly authorized by the national code of medical ethics.

(b) Taking part in any plan of medical care in which the doctor does not have professional independence.

(c) To receive any money in connection with services rendered to a patient other than the acceptance of a proper professional fee, or to pay any money in the same circumstances without the knowledge of the patient.

Under no circumstances is a doctor permitted to do anything that would weaken the physical or mental resistance of a human being, except from strictly therapeutic or prophylactic indications imposed in the interest of the patient.

A doctor is advised to use great caution in publishing discoveries. The same applies to methods of treatment whose value is not recognized by the profession.

When a doctor is called upon to give evidence or a certificate he should only state that which he can verify.

Duties of Doctors to the Sick

A doctor must always bear in mind the importance of preserving human life from the time of conception until death.

A doctor owes to his patient complete loyalty and all the resources of his science. Whenever an examination or treatment is beyond his capacity he should summon another doctor who has the necessary ability.

A doctor owes to his patient absolute secrecy on all which has been confided to him or which he knows because of the confidence entrusted to him.

A doctor must give the necessary treatment in emergency, unless he is assured that it can and will be given by others.

Duties of Doctors to Each Other

A doctor ought to behave to his colleagues as he would have them behave to him.

A doctor must not entice patients from his colleagues.

A doctor must observe the principles of "The Declaration of Geneva" approved by The World Medical Association.

DECLARATION OF GENEVA

Adopted by the General Assembly of The World Medical Association at Geneva, Switzerland, September, 1948.

At the time of being admitted as Member of the Medical Profession

I solemnly pledge myself to consecrate my life to the service of humanity.

I will give to my teachers the respect and gratitude which is their due;

I will practice my profession with conscience and dignity;

The health of my patient will be my first consideration:

I will respect the secrets which are confided in me;

I will maintain by all the means in my power, the honor and the noble traditions of the medical profession;

My colleagues will be my brothers;

I will not permit considerations of religion, nationality, race, party politics or social standing to intervene between my duty and my patient;

I will maintain the utmost respect for human life, from the time of conception; even under threat, I will not use my medical knowledge contrary to the laws of humanity.

I make these promises solemnly, freely and upon my honor.

These principles and this oath will answer for many physicians questions of conduct which occur frequently in medical practice. Here are the answers to such questions as euthanasia, advertising and soliciting of patients, consultation with cultists, publication of discoveries, the right of the patient to secrecy, and many similar problems. This code and this vow make clear that the one law which the physician must obey above all others is the law of humanity.

MORRIS FISHBEIN

MENTAL DISEASE AND MUSICAL STYLE

An extraordinary study made in a mental hospital in Surrey, England, concerned musical styles and mental disorders. Ten of the disturbed people, mostly young or middle aged adults, both men and women, were studied as to the effects upon them of a variety of musical selections played from records. Classical, romantic, impressionistic, realistic, and contemporary forms of music were tested, also music of the Far East, comical music, and folk songs.

The music of Bach, Mozart, and Beethoven failed to appeal to the mentally disturbed and rarely produced personal associations. Romantic music, including some of Brahms and Chopin, produced emotional reactions and personal associations were common. Some patients were left temporarily worse than previously by exposure to romantic music. Schizophrenics felt that they were unpleasantly stirred and stimulated to a feeling of failure by Tschaikowsky's Fifth Symphony. Impressionism had little effect.

Reactions to opera, such as "Carmen" and "Madam Butterfly" brought varying responses after the plots were explained, these responses indicating the personal problems of the mentally disturbed people who gave them. Strongly rhythmical march music with primitive features had the power to awaken a note of recognition among psychotics, but modern dance music had little effect. Folk songs particularly appealed and seemed to have a definite sublimating and integrating effect. As a form of treatment the playing of folk songs seemed to be especially useful for unbalanced people who craved security. The suggestion has been offered of possible value of music preferences among musicians as a form of psychological testing.

MORRIS FISHBEIN

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The Medical Bookman

PROCEEDINGS OF THE FIRST CLINICAL ACTH CONFERENCE*

On October 21 and 22, 1949, Armour & Company, Chicago, called a conference at the Hotel Stevens to discuss the status of the use of ACTH in a variety of diseases. In the Fall of 1946, the Armour company had supplied samples of the Adrenocorticotropin to Dr. George W. Thorn and his associates. Subsequently Dr. Fuller Albright confirmed the observations made by the first group. Thereafter the research was intensified and widened so that the report of this conference defines the status of our knowledge of the clinical use of ACTH until December 1, 1949.

The mere enumeration of the conditions benefited by this product is startling. From myasthenia and gout, studies were extended to nephrosis, rheumatoid arthritis, and rheumatic fever. More than two hundred investigators participated in the conference; fifty-two papers were presented and numerous discussions followed most of the presentations. Three weeks were required to transcribe the notes. Then it was determined that the importance of the material demanded prompt publication and circulation. The book of more than 600 pages and 400 pictures was available six weeks after this decision was made, the speed of publication itself setting a mark in medical publication worthy of the significance of the subject.

This pioneer volume indicates the manner in which the eosinophil count demonstrates the effectiveness of ACTH. The technic of the test is described clearly. The response of the human metabolism to ACTH has been determined at ages from infancy to senility. The effects of the product in cases of hypertension and in leukemia, in lupus erythematosus and periarteritis nodosa, and in all conditions affecting the collagen, are also amazing. Thus, it is not surprising that the Selye concept of stress and adaptation, supported by the work with ACTH and with Cortisone, should be hailed as a new era in medicine.

*Proceedings of the First Clinical ACTH Conference. Edited by John R. Mote, M.D., Medical Director, Armour Laboratories. 178 contributors. 607 pages, with 414 illustrations. 1950, The Blakiston Company, Philadelphia & Toronto. $5.50.

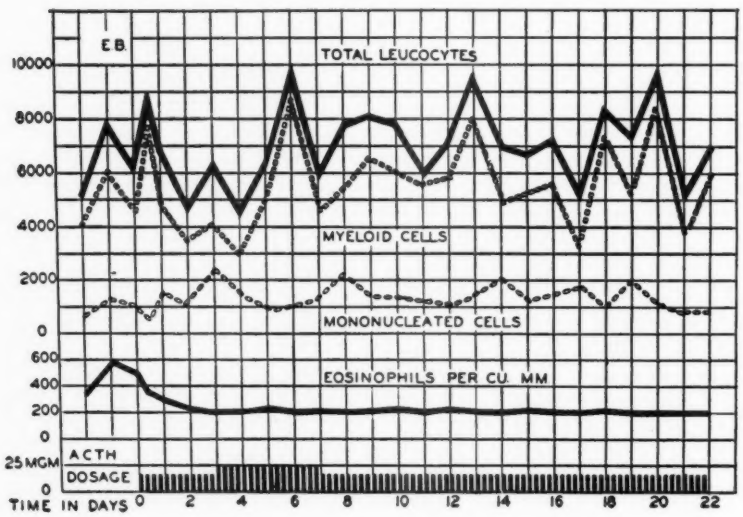

FIGURE 7 *(Page 5)*. Typical delayed eosinophil response of a patient with rheumatoid arthritis.

FIGURE 1a and b *(Page 319)*. Follicular lymphosarcoma: 100 mg. of ACTH daily for ten days.

(Illustrations from: Proceedings of the First Clinical ACTH Conference.)

A variety of glandular disturbances seem to benefit by the use of the adrenocorticotropic hormone, but failures have been recorded in several conditions, as well as failure in poliomyelitis and cancer in general. However, vast improvement in several personality disorders, in mental depression, and in delirium tremens again throws the balance far over on the favorable side. Studies are being extended now to the muscle and demyelinating diseases and leads of great importance are likely to be found in this clinical research.

The speed and intensity of modern medical research may, as shown by this book, seriously affect the questions of publication. Valuable time and many unnecessary losses of life would have occurred if the manuscripts, here assembled in book form, had drifted gradually into medical periodicals over a number of years. That was the plan followed in the past. The assembling of well controlled data and prompt reference to the medical profession is encouraging to the investigator and exceedingly useful to the physician who wishes to keep abreast of progress. When leaders can assemble and exchange ideas and information they save unnecessary repetition of basic studies. By such publication as is apparent in this book the value of the conference is enhanced thousands of times.

ALONG with announcement of clinical results with ACTH have come the usual reactions to therapeutic progress in medicine. People who have neither seen nor studied the work have voiced alarm concerning side-effects; one science writer, apparently quite unfamiliar with biology, declared that ACTH is a hoax; three or four reputable pharmaceutical manufacturers have launched alternative treatments and urged their use on the basis of a few poorly controlled and incompletely reported cases; several fly-by-night agencies have offered ACTH, untested, unstandardized, and possibly not even ACTH. In the meantime intensive research toward development of a synthetic product is under way and seems promising. The National Institute of Health is to have some millions for research on the subject.

A careful study of the scientific reports of the first clinical ACTH conference demonstrates the difference between the high level on which this work has been conducted and the carelessly organized, inadequately controlled studies that are being carried on outside the cooperative project. On the basis of some newspaper reports some are saying that ACTH produces diabetes; during the discussion of carbohydrate metabolism in the first clinical conference, Dr. George W. Thorn said: "In our experience, diabetes is not something we have had to worry about with ACTH administration over a prolonged period in a wide variety of diseases."

Dr. John R. Mote, medical director of Armour Laboratories, has contributed greatly to medical advancement by the production of this volume. His summarizing statement covers 613 patients studied over a period of three years. The work has only just begun but the possibilities indicated are stupendous. Physicians who want to participate in investigating ACTH should begin with this book.

M. F.

New Books Received

► THE ARTHROPATHIES; A Handbook of Roentgen Diagnosis - - -

By Alfred A. de Lorimier, M.D., Radiologist, Saint Francis Hospital, San Francisco; Consultant in Radiology for the United States Army at Letterman General Hospital; Formerly Commandant, The Army School of Roentgenology. Ed. 2. 335 pages, illustrated. 1949, The Year Book Publishers, Inc., Chicago. $7.00.

In this revised edition, the author has included a knowledge of developmental anatomy, detailed anatomic relationships, pathologic changes and a clinical understanding of disease entities which he illustrates with superb radiographic interpretation.

Main topics of the book are divided into a study of the peripheral joints, the joints of the spine, and the temporo-mandibular joints. A classification based on the fundamental etiology of the specific entity is utilized, and although cognizant that this classification has some shortcomings, the author considers it more reliable, as certain visible criteria tend to direct the examiner to include numerous diagnostic possibilities and one does not attempt to "read into" the x-ray findings that are present in a single entity. Bacteriologic, pathologic, and morphologic classifications originally considered were found to have certain disadvantages.

The book is not an atlas, but rather a textbook for accurate interpretation of roentgenologic findings in the joint regions. Each chapter includes criteria found in the soft tissues, the regional bones, articular cortexes, joint spaces in both early and late stages of the particular disease, and is followed by corroborative x-ray criteria, a roentgen summary, and finally differential considerations. The latter portion lists various diseases with similar x-ray findings and their differential criteria from the disease outlined.

In some chapters pertinent histories, physical findings, laboratory studies, and clinical course of the disease entity are helpful when the x-ray findings are not conclusive.

Since accurate diagnosis of early lesions requires visualization of adjacent soft tissues, certain technical factors are discussed which assist in delineating the contrasting media. With proper radiographic technic, the author feels it is possible to study nearly all tissues in and around joints. Illustrations representing the contrasting shadows brought on by variations in the kilovoltage, uses of casettes and intensifying screens, developing factors, and pneumoarthrography are impressive. It is emphasized that the entire study is a true scientific analysis of tangible evidence, an accumulation of both positive and negative findings, and not merely a "picture" of the condition. With careful consideration and good judgment a reliable conclusion can be achieved.

H. O'P.

► HUMAN PATHOLOGY - - -

By Howard T. Karsner, M.D., L.L.D., Former Professor of Pathology, Western Reserve University; Medical Research Advisor to the Bureau of Medicine and Surgery, United States Navy. Ed. 7. 927 pages, with 562 illustrations and 14 color plates. 1949, J. B. Lippincott Company, Philadelphia, London & Montreal. $12.00.

The new seventh edition of Karsner's text provides an excellent revision of this well known book. In it he has added two new chapters on the diseases of the eye and of the skin, and in addition, has revised or rewritten almost all of the other chapters to bring the material up to date.

The arrangement of subjects is similar to previous editions and is entirely satisfactory. This book is a well-written and easily-read complete text of general pathology. A large group of new photographs is excellent, and many of the drawings, both old and new, serve well to emphasize the material described verbally. It is unfortunate, however, that a few of the older drawings were not replaced.

The pathogenic approach employed by the author should prove of great value to the reader in understanding the ultimate pathologic picture of disease. Dr. Karsner's discussion of physiology and bacteriology as applied to pathology is helpful to clinician and pathologist alike in attaining a clear understanding of disease. A generous list of references to source material follows each chapter and provides a ready source of amplification of the textbook material.

S. S. B.

► BONE AND JOINT RADIOLOGY - - -

By Emerik Markovits, M.D., Formerly Scientific Collaborator of the Central Radiologic Institute of the General Hospital (Holzknecht-Institute), Vienna; Head of the Radiologic Department of Elizabeth Hospital of the City of Budapest; Post-graduate Lecturer at the Central Radiologic Institute of the University of Budapest; Radiologist at the Steiner Cancer Clinic, Atlanta, Georgia. 446 pages, with 616 figures. 1949, The Macmillan Company, New York. $20.00.

This well arranged and well illustrated text is an excellent guide to the early student in radiologic diagnosis. The first half of the book concerns itself with a general discussion of diseases of bone. The latter portion of the book describes bone and joint lesions of the skull, spine, and extremities.

The illustrations are well chosen and excellently reproduced. Many fine schematic sketches aid in explaining the pathologic processes. However, the illustrations are printed as either positive or negative prints which results in confusion to the reader.

The tabular forms of differential diagnosis should appeal greatly to the student who will obtain a satisfactory basis for analyzing the films and the case. The explanatory details in the text are concise but in parts rather brief. For this reason a practicing radiologist will need more extensive and detailed material to evaluate completely problem cases. For example, although injuries of the nucleus pulposus of the spine are concisely outlined, one paragraph describes the myelographic findings in disk protrusion. Obviously this is insufficient material to help the radiologist in this frequently occurring disability.

J. F.

► THE DIAGNOSIS AND TREATMENT OF ADRENAL INSUFFICIENCY - - -

By George W. Thorn, M.D., Hersey Professor of the Theory and Practice of Physic, Harvard Medical School; Physician-in-Chief, Peter Bent Brigham Hospital, Boston. With the collaboration of Peter H. Forsham, M.D., M.A. (Cantab.), Assistant in Medicine, Harvard Medical School and Peter Bent Brigham Hospital; and Kendall Emerson, Jr., M.D., Associate in Medicine, Harvard Medical School and Senior Associate in Medicine, Peter Bent Brigham Hospital. 171 pages, with 31 figures. 1949, Charles C Thomas, Springfield, Illinois. $5.50.

Dr. Thorn and his collaborators have written a helpful and timely monograph during this current period of the ascendency of Cortisone and ACTH. Following a brief historical introduction dealing with the development of adrenal cortical replacement therapy, the authors present concise material on the physiologic and chemical considerations. A complete differentiation between the "salt-retaining" and "carbohydrate-sparing" actions of the commercial preparations is made.

A chapter is devoted to discussions of the types of acute and chronic insufficiency, followed by a consideration of the symptoms of Addison's disease and laboratory findings, including EKG, x-rays, and electroencephalography. A valuable chapter is that dealing with the screening and tolerance tests in which the procedures are elaborated in detail.

The authors include a very practical outline of a diagnostic program with a listing of a five-day hospital testing period. An exhaustive chapter is devoted to treatment of chronic adrenal cortical insufficiency with special attention to complicating features. An interesting and complete discussion covers pellet-implantation therapy. Chapters dealing with differential diagnosis, treatment of adrenal crisis, toxic reactions, patients undergoing surgery, and prognosis, are contained in the volume. Throughout, there is emphasis on practical aspects—cost of therapy, facility of diagnosis, and analysis of the commercial products.

N. S.

► ACUTE LARYNGOTRACHEOBRONCHITIS - - -

By A. Harry Neffson, M.D. 197 pages, illustrated. 1949, Grune & Stratton, New York. $5.00.

In olden days many infections of the lower airways were designated as "croup." This appellation applied to any disability in the larynx which obstructed ingress and egress of air. Some were

"membranous"; others had no demonstrable membrane. Those with membrane were often "diphtheritic" and were occasionally misdiagnosed, but some nondiphtheritic infections also give a membrane to which the term "pseudo" was applied. Before O'Dwyer's intubation, many children with croup died promptly. This life-saving instrumentation marked a new era in laryngeal obstructions.

Dr. Neffson has treated some 2,000 cases of glottis blockage, most of them successfully. He relies on direct suction-laryngoscopy, intubation, or, finally, on tracheotomy. He thinks that the latter is seldom necessary. It is beset with greatly increased secretions, gumming and plugging, and blocking of the tube, removal of the tube by obstreporous patients, and surgical shock, with ultimate scarring or strictures, etc.

There are disadvantages in any surgical undertaking. Tracheotomy is the easiest of all procedures for the average physician to perform, and is definitely life-saving and immediate. The pity is that it is often undertaken too late! Undue conservatism in trying out other measures first may be dangerous to life, and often is.

I. W. V.

► A TEXTBOOK OF NEUROPATHOLOGY - - -

By Ben W. Lichtenstein, B.S., M.S., M.D., Associate Professor of Neurology, The University of Illinois College of Medicine; State Neuropathologist, Illinois Neuropsychiatric Institute. New, 1st Edition. 474 pages, with 282 figures. 1949, W. B. Saunders Company, Philadelphia & London. $9.50.

The author has admirably condensed into this text the ever-widening field of neuropathology. It is written primarily for the medical student and for those training in neurology, psychiatry, pathology, and neurologic surgery. It will nevertheless prove most useful to the general pathologist who finds that he is called upon more frequently for pathologic diagnoses of diseases of the central nervous system. The neurosurgeon also will find this text most helpful in reviewing many neurologic conditions which he may see infrequently, but a knowledge of which he must have for his differential diagnoses.

One of the many attributes of this text is that it contains all the essential information that a student should have regarding the neuropathology of the different diseases. Where there are controversies regarding the etiology of a disease the more prominent theories are mentioned and no attempt is made to be dogmatic. Each chapter is followed by numerous references so that the student can amplify the text which must necessarily be at times brief.

A correlation of the clinical and pathologic picture is most difficult to obtain in any textbook, be it medical, surgical, or pathologic, but in this instance the author has seen fit to include enough clinical material so that there is an excellent balance between the two. The illustrations add much to the text and are informative.

W. P. R.

► CYSTOSCOPY AND UROGRAPHY - - -

By James B. Macalpine, D.Sc. (Honoris Causa), F.R.C.S. (Eng.), Lately Honorary Surgeon and Surgeon in Charge of the Genitourinary Department, Salford Royal Hospital; Consulting Urologist, Christie Hospital and Holt Radium Institute, Manchester. Ed. 3, revised and enlarged. 554 pages, with 338 illustrations and 15 colored plates. 1949, The Williams and Wilkins Company, Baltimore. $13.50.

This book covers considerably more territory than one would suspect from its title. In addition to the subjects mentioned in the title it deals with various types of operative cystoscopy, including transurethral resection, discusses tests of renal function at some length, and goes into the chemistry and behavior in the body of the various compounds used for excretory urography.

Because it deals rather extensively with fundamentals, this book is really most useful for the beginner at cystoscopy and for the practitioner who performs cystoscopic examinations occasionally, but who does not make it a major interest. It presents in considerable detail the history, construction, and optical characteristics of cystoscopes as well as their sterilization and maintenance. Anesthesia, the routine study of urologic patients, the cystoscopic and pyelographic findings, and almost any disorder that one can recall are dealt with in a satisfactory manner. A chapter on the pathogenesis of renal tuberculosis has been inserted rather apologetically.

Like most British books dealing with Urology,

it has a bibliography which is quite scanty in relation to the size and scope of the work. It is amusing to note that the average British cystoscope apparently does not have a valve which permits irrigation of the bladder while inspection is being carried out, and that the author overlooks the fact that, given a proper cystoscope, satisfactory examination can usually be made despite vigorous bleeding. He also ignores the fact that clots can almost invariably be evacuated through the cystoscope so as to permit the examination to go forward at once, because he states that one may have to wait many days for clots to disappear in order to permit a satisfactory examination.

Despite these minor faults, the book is recommended to urologic residents and to general practitioners who do cystoscopies occasionally. It will repay reading by the accomplished cystoscopist as well.

C. D. C.

Books received will be acknowledged in this department each month. As space permits, books of principal interest to our readers will be reviewed more extensively.

Postgraduate Gastroenterology. Edited by Henry L. Bockus, M.D., Professor of Gastroenterology, University of Pennsylvania Graduate School of Medicine. 670 pages, with 258 figures. 1950, W. B. Saunders Company, Philadelphia & London. $10.00.

Applied Medicine. By G. E. Beaumont, M.A., D.M., F.R.C.P., D.P.H., Physician to the Middlesex Hospital and Hospital for Diseases of the Chest, Brompton. 540 pages, with 74 illustrations. 1950, The Blakiston Company, Philadelphia & Toronto. $6.00.

Practical Statistics in Health and Medical Work. By Ruth Rice Puffer, Dr. P.H., Director of Statistical Service, Tennessee Department of Public Health; Department of Preventive Medicine and Public Health, Vanderbilt University School of Medicine. Foreword by Hugh Muench, M.D., Professor of Biostatistics, Harvard University School of Public Health. 238 pages. 1950, McGraw-Hill Book Company, Inc., New York, Toronto & London. $3.75.

Urological Surgery. By Austin Ingram Dodson, M.D., F.A.C.S., Professor of Urology, Medical College of Virginia; Urologist to the Hospital Division, Medical College of Virginia; Urologist to Crippled Children's Hospital; Urologist to St. Elizabeth's Hospital; Urologist to St. Luke's Hospital and McGuire Clinic, Richmond, Virginia. Ed. 2. 855 pages, with 645 illustrations. 1950, The C. V. Mosby Company, St. Louis. $13.50.

Penicillin; Its Practical Application. Under the General Editorship of Sir Alexander Fleming, M.B., B.S., F.R.C.P., F.R.C.S., F.R.S., Professor Emeritus of Bacteriology, University of London; Principal, Wright-Fleming Institute of Microbiology, St. Mary's Hospital Medical School, London. Ed. 2. 491 pages, with 63 figures. 1950, Butterworth & Co. (Publishers) Ltd., London, and The C. V. Mosby Company, St. Louis. $7.00.

Introduction to Neuropathology. By Samuel Pendleton Hicks, M.D., Departments of Pathology, Harvard Medical School and the New England Deaconess Hospital; Formerly Consultant in Neuropathology, National Naval Medical Center; and Shields Warren, M.D., Departments of Pathology, Harvard Medical School and the New England Deaconess Hospital; Director, Division of Biology and Medicine, U. S. Atomic Energy Commission. First edition. 494 pages, illustrated. 1950, McGraw-Hill Book Company, Inc., New York, Toronto & London. $10.00.

1950 Current Therapy; Latest Approved Methods of Treatment for the Practicing Physician. Editor: Howard F. Conn, M.D. Consulting Editors: M. Edward Davis, M.D., Vincent J. Derbes, M.D., Garfield G. Duncan, M.D., Hugh J. Jewett, M.D., William J. Kerr, M.D., Perrin H. Long, M.D., H. Houston Merritt, M.D., Paul A. O'Leary, M.D., Walter L. Palmer, M.D., Hobart A. Reimann, M.D., Cyrus C. Sturgis, M.D., and Robert H. Williams, M.D. 736 pages. 1950, W. B. Saunders Company, Philadelphia & London. $10.00.

A Manual of Cardiology. By Thomas J. Dry, M.A., M.B., Ch.B., M.S. in Medicine; Associate Professor of Medicine, University of Minnesota (Mayo Foundation); Consultant in Section on Cardiology, Mayo Clinic. New, 2nd edition. 355 pages, with 97 figures. 1950, W. B. Saunders Company, Philadelphia & London. $5.00.

Medical Gynecology. By James C. Janney, M.D., F.A.C.S., Associate Professor of Gynecology, Boston University School of Medicine; Associate Visiting Gynecologist, Massachusetts Memorial Hospital. New, 2nd edition. 454 pages, with 108 figures. 1950, W. B. Saunders Company, Philadelphia & London. $6.50.

Handbook of Obstetrics and Diagnostic Gynecology. By Leo Doyle, M.S., M.D. First edition. Illustrations by Ralph Sweet. 240 pages. 1950, University Medical Publishers, Palo Alto, California. $2.00.

The Cytologic Diagnosis of Cancer. By the staff of the Vincent Memorial Laboratory of the Vincent Memorial Hospital, a gynecologic service affiliated with the Massachusetts General Hospital, Boston, Mass. The Department of Gynecology, Harvard Medical School. Published under the sponsorship of the American Cancer Society. 229 pages, with 153 figures. 1950, W. B. Saunders Company, Philadelphia & London. $6.50.

Training for Childbirth; A Program of Natural Childbirth with Rooming-In. By Herbert Thoms, M.D., Professor of Obstetrics and Gynecology, Yale University School of Medicine; Obstetrician and Gynecologist-in-Chief, Grace-New Haven Community Hospital, University Service. 114 pages, illustrated. 1950, McGraw-Hill Book Co., Inc., New York, Toronto & London. $3.00.

Medical Management of Gastrointestinal Disorders. By Garnett Cheney, M.D., Clinical Professor of Medicine, Stanford University Medical School. 478 pages, illustrated. 1950, The Year Book Publishers, Inc., Chicago. $6.75.

A Guide to General Medical Practice. By Martin G. Vorhaus, M.D., Attending Physician, Hospital for Joint Diseases, New York City. 244 pages. 1950, The Macmillan Company, New York. $3.50.

Consultation Service

This special consultation information service is offered as a regular monthly feature of *Postgraduate Medicine*. Readers are invited to call on this Service for answers to difficult medical problems from our consultants and members of our Editorial Board best qualified to help. Each question will be answered by mail and those of general interest will be published each month. Address all communications to Consultation Service, *Postgraduate Medicine*, 516 Essex Building, Minneapolis 3, Minnesota.

CHEYNE-STOKES RESPIRATION

QUESTION: Present complaint: Irregular respiration in normal man, 63 years of age, upholsterer. About 5 to 7 breaths, beginning shallow, increasing in intensity, then decreasing and finally ceasing, followed by apnea of about the same length of time as respirations.

There are no other complaints. Heart action and kidney function normal. Metabolism about −24, difficult to take because of respiration. There is a marked stuffiness of nose and head coming on in the evening and night. Submucous resection and local treatments have given no relief.

M.D.—Maryland

ANSWER: From the description of this type of breathing, it is Cheyne-Stokes in character. It is seen occasionally in very young and in elderly people without any actual disease. In the patient described I think it would be fairly safe to assume that the patient has moderate arteriosclerosis affecting the blood vessels which supply the medulla, and that the type of breathing is most likely due to senile degenerative changes in this part of the central nervous system.

It is well known that this type of breathing occurs in such pathologic conditions as uremia, chronic heart failure, narcotic poisoning, pathologic softening of the brain, and in certain lesions of the brain such as tumors, hydrocephalus, etc. The stuffiness of the nose and head which comes on in the evening and night is probably on an allergic basis, and one would not expect submucous resection or local treatment to give relief in a Cheyne-Stokes respiration. The possibility of allergy should be investigated as the cause of this stuffiness.

JAMES E. PAULLIN

TREATMENT OF MULTIPLE SCLEROSIS

QUESTION: Several patients with multiple sclerosis have inquired lately concerning the reported cure of this disease in one or two instances during the last few months. I believe the reports came from the Kaiser Institute in California. Can you give me any information along these lines?

M.D.—Wisconsin

ANSWER: As long as multiple sclerosis is not a verified diagnosis by definite, positive laboratory findings, one can only assume clinically that we are dealing with a disease syndrome which we formulate into the clinical term multiple sclerosis.

It is a well known fact that multiple sclerosis with and without therapy may have long-lasting remissions during which the patient is subjectively and quite commonly also clinically objectively free.

I do not believe that anybody is justified to report a cure in multiple sclerosis since we do not know the etiologies of, nor any specific remedies for multiple sclerosis. There are no doubt clinical syndromes which resemble multiple sclerosis even to the specialist. However, such disorders are grouped (1) infectious (virus) encephalomyelitis; (2) under neuro-allergy. If the allergic sensitizing factors are eliminated the clinical symptoms very much like those of multiple sclerosis will disappear. Nevertheless, the disposition to new allergic irritation is present, and the transient clinical freedom is akin to sufferings of a hay fever candidate.

It is my opinion that one should not speak today of a cure for multiple sclerosis. I request to call it a clinical remission with freedom from objective and subjective manifestations. The Kaiser-Kabat Institutions do not have a "specific remedy" against multiple sclerosis. They are treating this disorder with rest, general diversified support procedures but mainly and quite effectively by re-educational physical means.

HANS H. REESE

CURARE IN TREATMENT OF TETANUS

QUESTION: May curare safely be used in the treatment of tetanus in children, and in what dosage? Does it in any way interfere with the action of tetanus antitoxin?

M.D.—Missouri

ANSWER: Curare is a dangerous drug. While theoretically ideal for control of the severe and painful muscular spasms of tetanus, its prolonged use is difficult and dangerous. Excessive amounts may result in circulatory as well as respiratory collapse. With complete relaxation the tongue tends to fall back, blocking the pharynx. This coupled with the hypopharyngeal secretions present, gives rise to difficulty in keeping the airway clear.

Thus the use of this drug requires the constant attention of a trained personnel whether it be used in the multiple dose or prolonged method. When used as an aqueous solution the recommended dose is one-half unit per pound of body weight given every three to four hours as needed. The effects of the drug may be counteracted by prostigmine. Artificial respiration may be needed while waiting for prostigmine to become effective.

Whatever the type of sedation used in the treatment of tetanus, early tracheotomy should be considered in all severe cases.

LEE FORREST HILL

TREATMENT OF NAUSEA DURING PREGNANCY

QUESTION: I have a patient who has had nausea and vomiting with each of four pregnancies, including the present one. She had it for the whole seven to eight months, beginning at the sixth week. The baby is very much wanted so I do not feel this is a "neurotic" case.

Basal metabolic rate has always been low (–2) but treated with 2 gr. of thyroid every day. She has previously had pyridoxine intravenously with temporary relief, diminishing with each injection.

Are there diagnosable causes or other treatment for this miserable condition?

M.D.—British Columbia

ANSWER: There is certainly more than appears on the surface of this brief history.

For prompt relief the use of Dramamine is recommended (50 mg. twice daily tapering off rapidly to use only as required).

Meantime a thorough review should be made of this patient's daily living, eating, and sleeping habits.

What about her household cares and can she be given some help, as by employment of a part-time day worker? Three children to look after are too much for this woman, and her other housework may be a heavy burden to her. Relatives do not usually make good substitutes for a paid helper, and in fact, the "in-laws" situation in this family should be scrutinized.

Does her husband smoke cigars or pipes in the house, and especially does he leave the "dead butts" or used pipes lying around for her to smell?

Coitus should be prohibited, at least for a time.

Her diet should be investigated. Carbohydrates should be increased, and to avoid an unbalancing of her diet if it was originally fairly well arranged, protein concentrates should be advised. Small meals at frequent intervals are preferable to three larger meals a day, and a definite schedule for these should be established. Bizarre items should be deleted from the diet. A chocolate malted milk shake or a chocolate almond bar, for examples, are not well-chosen meals.

Vitamin B_1 and B_6 deficiency must be combatted by the use of vitamin B complex. One Gravidox (Lederle) pill daily is effective.

With a basal metabolic rate so near to normal (–2) it is unlikely that the patient needs any thyroid substance, and two grains daily might create enough nervous tension to aggravate her nausea. One symptom of thyroid overdosage is nausea and vomiting.

Hospitalization seems advisable for this patient in order to stop her nausea and vomiting and then to establish a new regimen of living at home so that her illness will not persist this time throughout her pregnancy.

PAUL TITUS

SENSITIVITY TESTS FOR PENICILLIN AND SULFONAMIDES

QUESTION: Is there any satisfactory test that can be made to determine if a patient is allergic to penicillin and sulfonamide drugs—for the average case that comes to the general practitioner?

Is the water-soluble penicillin less likely to produce allergic reaction than the oil and wax preparations? Also, does the water-soluble preparation give as long a therapeutic benefit as the oil?

M.D.—Nebraska

ANSWER: It is possible to test for sensitivity to the spore of the mold Penicillium, but there are no reliable tests for sensitivity to any of the drugs. Touching the tongue with a suspected drug has been tried but is most unreliable. The reaction to penicillin is usually a delayed one and is characterized usually by urticaria or angio-edema and usually appears about two weeks after the drug was started.

The water-soluble penicillin is less likely to produce reactions because some individuals are sensitive either to the wax or oil used as the vehicle.

The water-soluble penicillin will maintain a sufficiently adequate level for twenty-four hours.

HARRY L. ROGERS

TREATMENT OF HYPERHIDROSIS

QUESTION: I have a female patient, aged 70, who has suffered from generalized hyperhidrosis for the past four years. She is somewhat anxious, but I can find no gross organic abnormality. She does not benefit from bromides, belladonna or from local applications.

I would be grateful for any advice you could offer me.

M.D.—Manitoba

ANSWER: Generalized hyperhidrosis is caused by overfunctioning of the sympathetic nervous system. If a hyperthyroid state has been ruled out, the disturbance probably is explained on a functional basis similar to that which exists in Raynaud's syndrome. For a patient 70 years old one would scarcely wish to consider such a radical procedure as sympathetic ganglionectomy. There is no adequate treatment which can be applied to the skin with any degree of satisfaction; the administration of phenobarbital by mouth, aided by psychotherapy, might be helpful. It is unusual for generalized hyperhidrosis to last four years.

PYELITIS OF PREGNANCY

QUESTION: Would chloromycetin and aureomycin be of any help in recurrent pyelitis of pregnancy?

M.D.—Connecticut

ANSWER: Chloromycetin is the preferred antibiotic in urinary tract infections. It is well tolerated and inhibits a wider range of organisms than aureomycin.

JOSEPH L. BAER

New Drugs *and* Instruments

Information published in this department has been supplied by the manufacturers of the products described.

AEROHALOR

PURPOSE: Reduction of bronchospasm in asthma.

DESCRIPTION: Powder inhaler and three Aerohalor cartridges containing 100 mg. each of Norisodrine sulfate powder 10 per cent for oral inhalation.

PRODUCER: Abbott Laboratories, North Chicago, Ill.

ORETON VIAL
100 mg. per cc.

PURPOSE: High dosage form of testosterone propionate for intramuscular administration of large amounts of the hormone where indicated.

COMPOSITION: Testosterone propionate, U.S.P., in sesame oil.

DOSAGE AND ADMINISTRATION: Palliation of breast carcinoma—100 mg. three times weekly by intramuscular injection.

HOW SUPPLIED: Multiple dose vials of 10 cc., containing 100 mg. per cc.

PRODUCER: Schering Corporation, Bloomfield, N. J.

KREISELMAN PORTABLE BELLOWS RESUSCITATOR

DESCRIPTION: Unit can be used efficiently in cases of respiratory failure, regardless of the patient's position, either with or without oxygen. Artificial respiration may be started immediately with air and, if necessary, oxygen may be added when available without interrupting the procedure.

The model 115 resuscitator consists of a bellows of about 1,600 cc. capacity, a face mask, and a valve placed between them. There are valves for intake of air and oxygen, and a safety valve which limits pressure to 20 millimeters of mercury. All valves operate automatically. Weight of the unit is two pounds.

PRODUCER: Ohio Chemical & Surgical Equipment Co., Madison, Wis.

NEOTRESAMIDE

PURPOSE: Triple sulfonamide solution for intravenous use.

COMPOSITION: Contains 2.4 per cent sodium sulfadiazine; 2.4 per cent sodium sulfamethazine; 1.2 per cent sodium sulfamerazine.

INDICATIONS FOR USE: Treatment of infections in which sulfamerazine, sulfadiazine, or sulfamethazine alone is indicated.

DOSAGE AND ADMINISTRATION: Approximately 0.05 gm. per kilogram of body weight should be administered as initial dose. Immediately after completion of intravenous injection, oral therapy should be started to maintain an effective level. Parenteral administration of the solution should be discontinued as soon as a satisfactory blood level can be maintained by oral administration.

HOW SUPPLIED: In packages of one ampule and in lots of 6, 25, and 100 ampules.

PRODUCER: Sharp & Dohme, Inc., Philadelphia, Pa.

PLURAVIT® DROPS

PURPOSE: Multiple vitamin liquid for prevention of vitamin deficiencies.

COMPOSITION: Each 0.6 cc. supplies:

Vitamin A	5,000 units
Vitamin D_2	1,000 units
Vitamin B_1	1 mg.
Vitamin B_2	0.4 mg.
Vitamin B_6	1 mg.
Nicotinamide	5 mg.
Pantothenic acid	2 mg.
Vitamin C	50 mg.

DOSAGE AND ADMINISTRATION: Best administered in orange juice in which it is unnoticeable. May be incorporated in cereals, puddings, and other foods. Daily prophylactic dose: For infants—0.3 cc.; older children and adults—0.6 cc. For pregnant and nursing women, 0.6 cc. two or three times daily. Therapeutic dose is approximately five times the daily prophylactic dose.

HOW SUPPLIED: Bottles of 15 cc., with a special dropper calibrated for 0.3 and 0.6 cc.

PRODUCER: Winthrop-Stearns, Inc., New York, N. Y.

ARTANE ELIXIR

PURPOSE: Convenient dosage form of Artane* Trihexyphenidyl, antispasmodic for treatment of parkinsonism.

COMPOSITION: Each teaspoonful contains 2 mg. of the active drug in a 5 per cent alcohol, cola-flavored liquid.

HOW SUPPLIED: In pint and gallon bottles.

PRODUCER: Lederle Laboratories Division, American Cyanamid Co., Pearl River, N. Y.

*Trademark.

(Continued on page A-50)